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Archives OF OTOLARYNGOLOGY

Chronic Bacterial Allergy of the Perinasal Sinuses
John R. Surber

Direct Adenoidectomy — *Paul Guggenheim*

Mobilization of the Stapes Without Incision
Mervin C. Myerson

Pharyngomaxillary-Space Abscess — *Layton S. Rogers*

Hypoadrenocorticism in Otolaryngology
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Labyrinthine and Intracranial Lesions
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The Cochleopalpebral Reflex in Normal Hearing and Hard of Hearing Persons
James P. Albrite, Robert A. Butler, and F. Thomas Galloway

A New Alcohol-Caloric Test for Vestibular Function
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Disposable Rhinoplastic Blades
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Otitis Media and Complications
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Complete Contents on First Inside Page

NOVEMBER 1956
VOLUME 64 NUMBER 5

From Surber, p. 351

AMERICAN MEDICAL ASSOCIATION Publication

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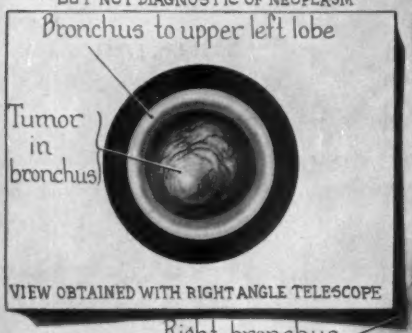

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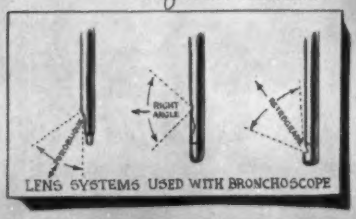

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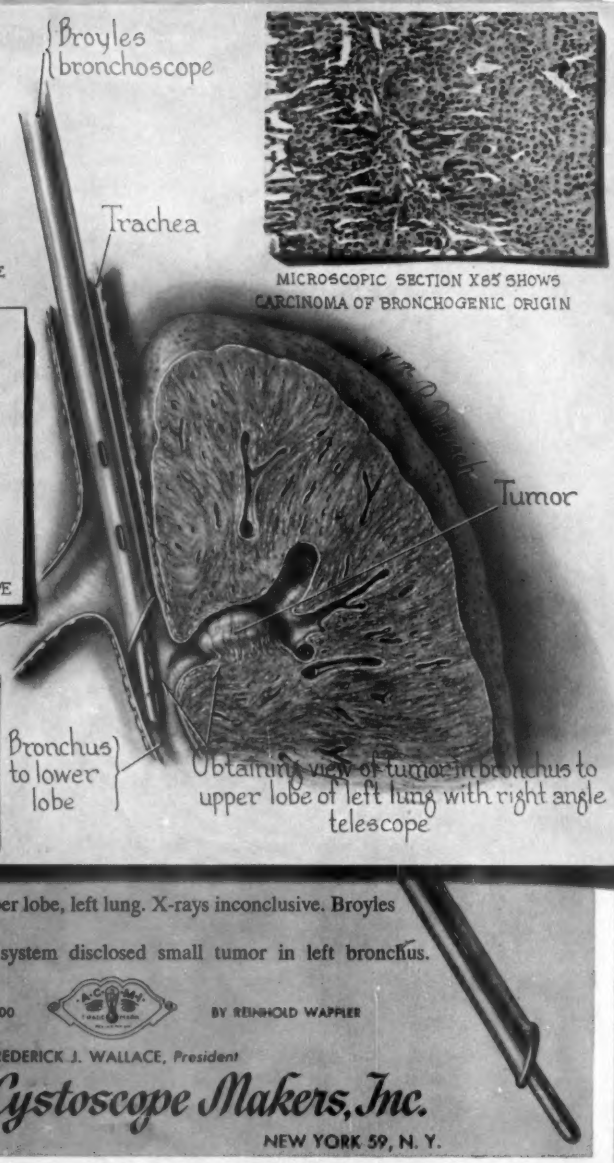

Obtaining view of tumor in bronchus to upper lobe of left lung with right angle telescope

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TABLE OF CONTENTS

VOLUME 64 NOVEMBER 1956 NUMBER 5

ORIGINAL ARTICLES

PAGE

Chronic Bacterial Allergy of the Perinasal Sinuses
John R. Surber, M.D., New York 351

Direct Adenoidectomy
Paul Guggenheim, M.D., Topeka, Kan. 361

Mobilization of the Stapes Without Incision
Mervin C. Myerson, M.D., Beverly Hills, Calif. 373

Pharyngomaxillary-Space Abscess
Layton S. Rogers, M.D., Whittier, Calif. 377

Hypoadrenocorticism in Otolaryngology
Herbert B. Goldman, M.D., Rockville Centre, N. Y., and John W. Tintera, M.D., Yonkers, N. Y. 381

Labyrinthine and Intracranial Lesions
Burech Rachlis, M.D., Philadelphia 390

AUDIOLOGY SECTION

The Cochleopalpebral Reflex in Normal Hearing and Hard of Hearing Persons
Major James P. Albrite (MC), U. S. Army; Robert A. Butler, Ph.D., Washington, D. C., and Pvt. F. Thomas Galloway, Medical Department, U. S. Army 402

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

A New Alcohol-Caloric Test for Vestibular Function
Herman I. Laff, M.D., and G. L. Smith, M.D., Denver 409

Disposable Rhinoplastic Blades
Nathan L. Fineberg, M.D., Boston 412

PROGRESS IN OTOLARYNGOLOGY

Otitis Media and Complications
B. R. Dysart, M.D., Pasadena, Calif. 413

REGULAR DEPARTMENTS

Abstracts from Current Literature 425
News and Comment 431
Directory of Otolaryngological Societies 433

SCIENTIFIC EXHIBITS

Cancer of the Larynx
Chevalier L. Jackson, M.D.; John V. Blady, M.D., and Charles M. Norris, M.D., Philadelphia 435

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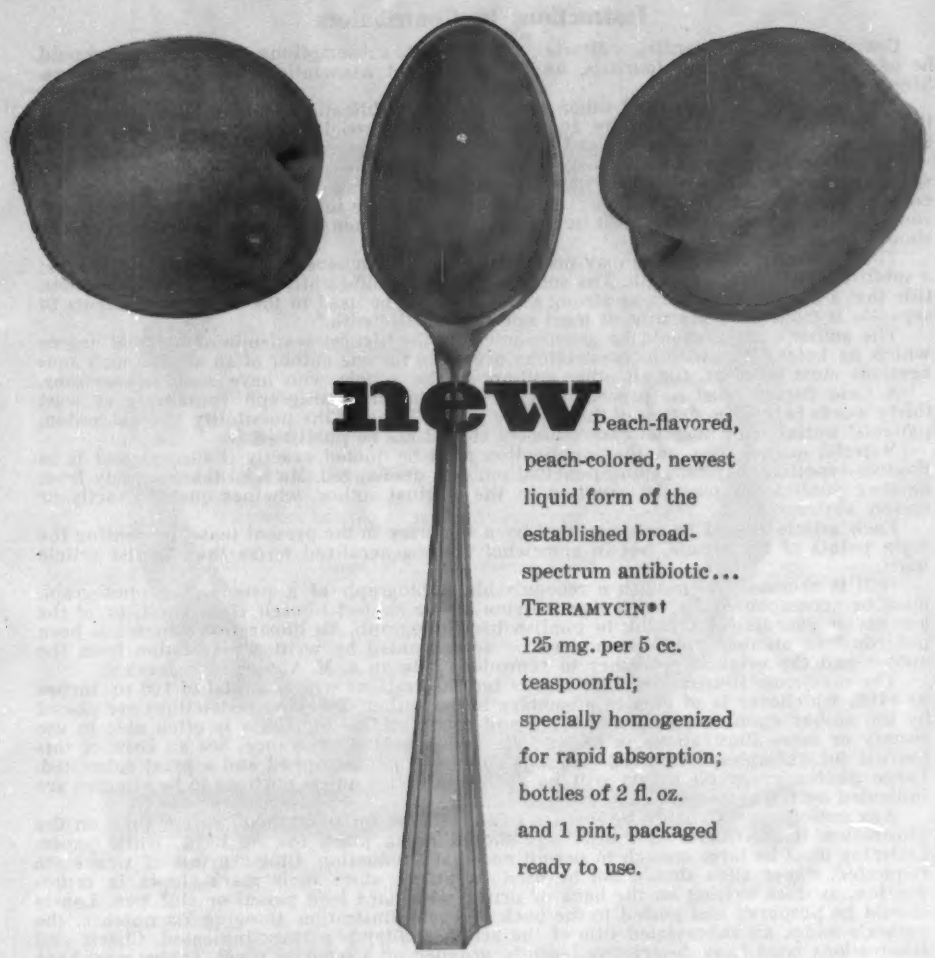

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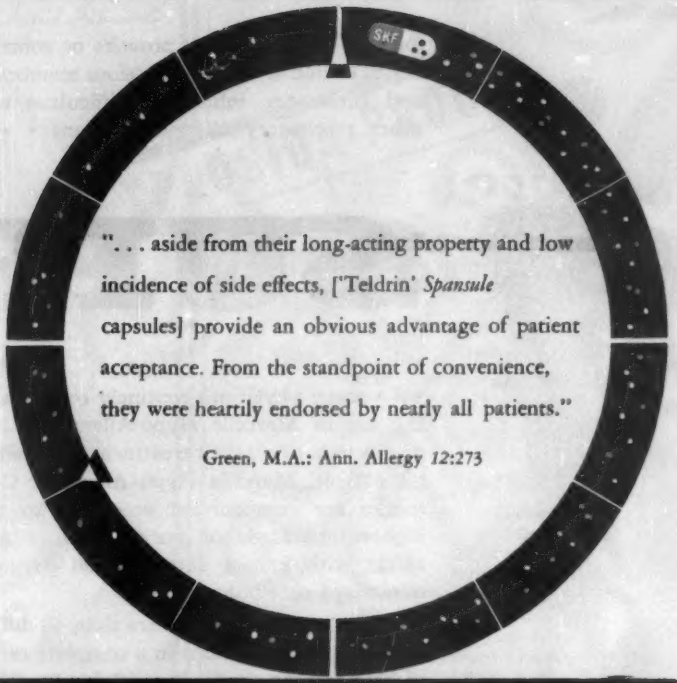

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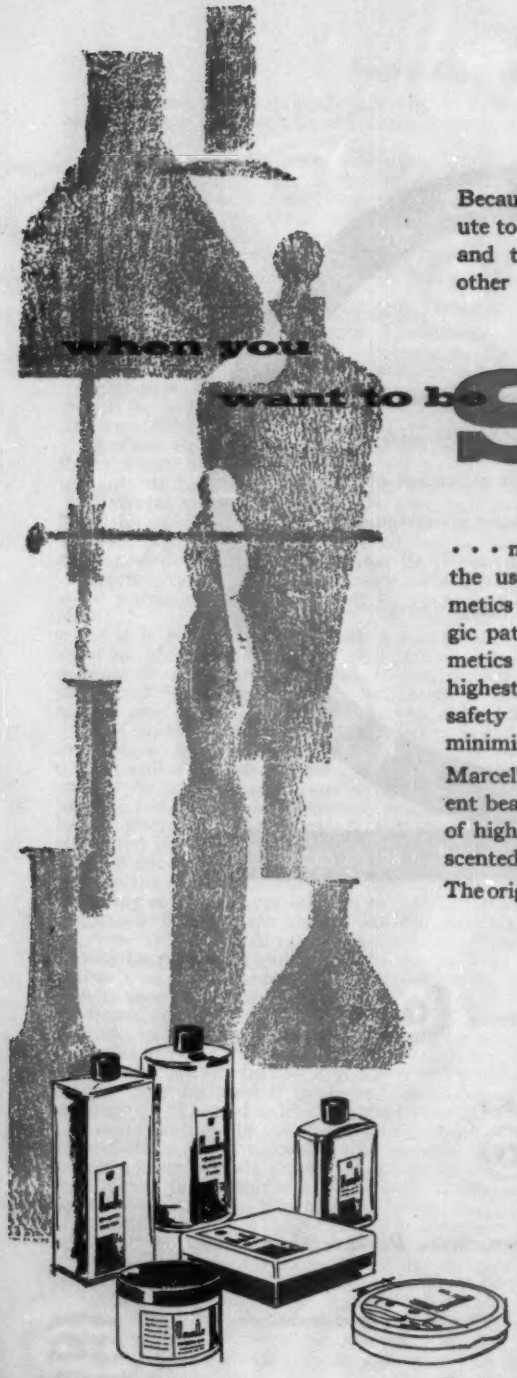

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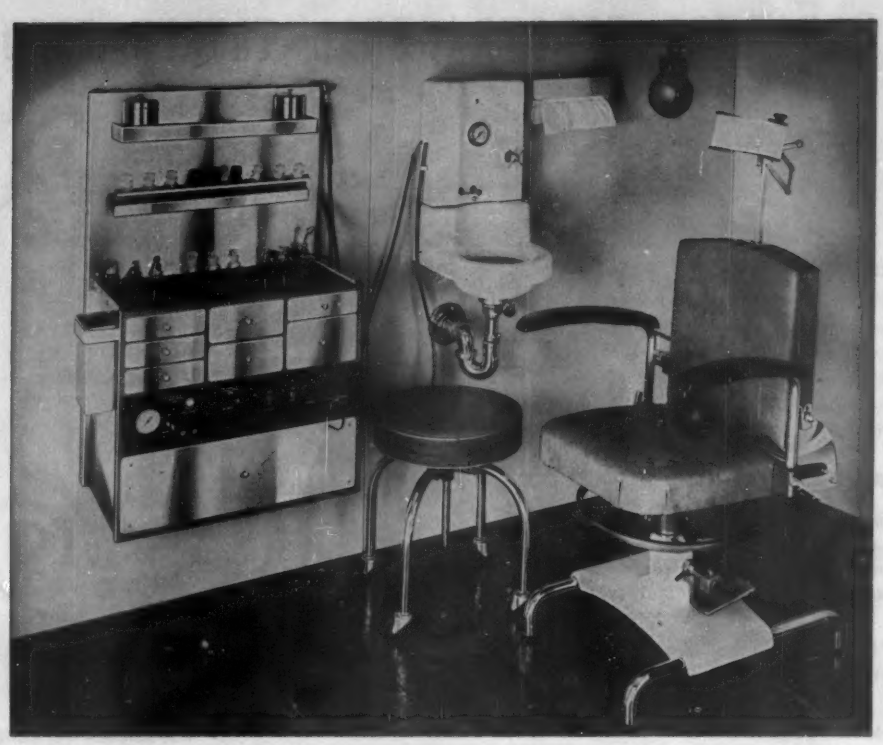

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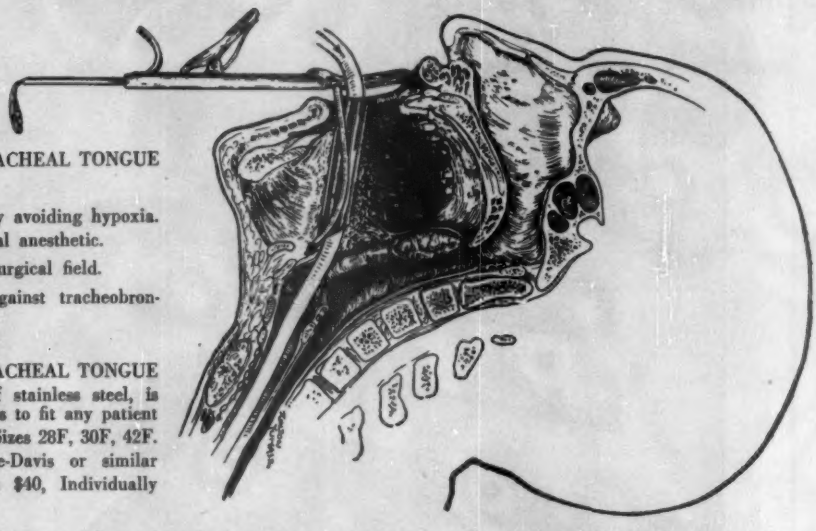

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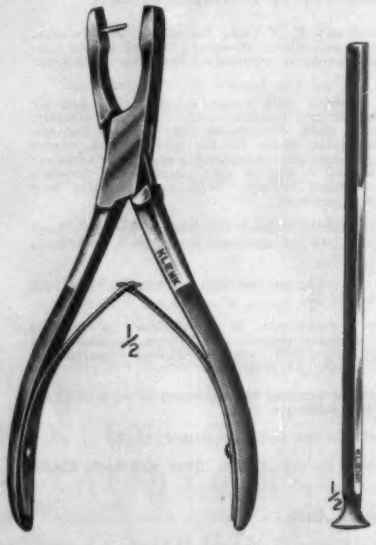

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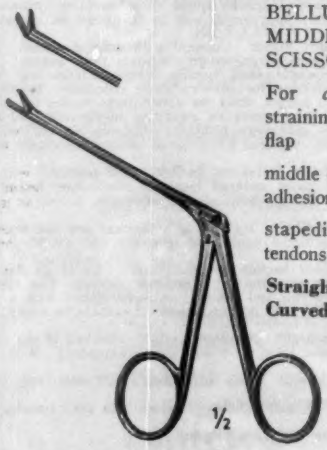

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Chronic Bacterial Allergy of the Perinasal Sinuses

A Report of Sixty-Five Cases

JOHN R. SURBER, M.D., New York

"A patient half as wretched with pulmonary tuberculosis would be put into a sanitarium."[1] Yet chronic bacterial sinusitis is even more insidious and frustrating than tuberculosis, since the patient frequently appears to be in good health and is regarded by his friends and associates as lazy and a procrastinator. One afflicted with this debilitating condition is often so depressed and miserable that he is unable to think, unable to compete in daily life.

Moreover, diagnosis and effective treatment are in many cases complicated by the fact that this disease may resemble other diseases or be concurrently present with them. Many conditions, such as arthritis, bronchiectasis, pulmonary tuberculosis, peptic ulcer, hypertension, and cholecystitis, may be severely aggravated by bacterial sinusitis. Again, the multiplicity of symptoms exhibited by these frustrated, chronically ill persons can further confuse and impair effective treatment.

Report of Cases

The following 14 cases, selected from the 65 on which this study is based, illustrate certain especially significant points. First, they suggest the variety of symptoms which are characteristic of this disease. Second, they present problems as to why some carefully diagnosed and treated patients respond poorly to every kind of therapy. Finally, before the cultures described in this paper were done, antibiotics had removed sensitive organisms to a large extent, and the cultures in this series usually disclosed strains of streptococci which are resistant to all antibiotics.

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From the Departments of Otolaryngology: St. Clare's Hospital and the Manhattan Eye, Ear and Throat Hospital.

CASE 1.—A 22-year-old woman was first seen in my office on Aug. 21, 1954. For two and a half years she had suffered from persistent sneezing, recurrent nasal blocking, and episodes of severe asthma. External allergens had been eliminated as probable causative factors. Roentgenograms of the sinuses (Fig. 1) did not indicate that there were pathologic conditions of the mucous membrane. However, cultures of nose and throat washings revealed the presence of Streptococcus salivarius and mitis and also showed that these organisms had strong reduction powers and were exceedingly toxic. Autogenous vaccines, with the use of the controls described later in this report, greatly reduced both the frequency and the severity of the asthmatic attacks and also eliminated the sneezing and nasal blocking except for occasional episodes.

CASE 2.—A male patient was first seen in my office on June 13, 1949. His chief complaints were intense weariness which was unrelieved by sleep, stuffiness in the ears, persistent wheezing cough, episodes of nasal blocking, and severe general malaise. During the preceding four years he had not been able to keep a job more than a few weeks at a time, and his friends considered him worth-

Fig. 1 (Case 1).—X-ray of sinuses showing little evidence of the chronic bacterial allergy causing severe asthmatic attacks during the past two and one-half years.

less. Roentgenograms showed a dense chronic thickening of the lining membranes of both maxillary sinuses, and a fluid level in the left one. Culture of nose and throat washings disclosed a viridans Streptococcus. In spite of attempts to control the symptoms by autogenous vaccines and antral irrigation, purulent washings remained consistently available from both maxillary sinuses. Therefore, on March 7, 1951, a bilateral Caldwell-Luc operation was performed. The symptoms diminished rapidly. Three months later the patient had obtained employment, and he has worked steadily ever since.

Case 3.—A 40-year-old man was first seen in my office on Dec. 4, 1951. Since 1946 he had suffered from increasingly severe malaise, general aching and pains, wheezing persistent cough, anorexia, nasal blocking, and dull fronto-occipital headache. He had been less and less able to carry out the duties of his job, and, in spite of a long record of faithful service, his friends believed him neurotic. For one year before he came to my office he had been receiving dust extracts and other forms of treatment, but without relief. Roentgenograms showed a severe pansinusitis (Fig. 2), and culture of nose and throat washings disclosed a viridans Streptococcus. Because the sinuses were so extensively involved, radical operation seemed imperative. Accordingly, on Jan. 6, 1952, a submucous resection and transantral ethmoidectomy were done. Massive doses of chloramphenicol (Chloromycetin), to which the organism is believed sensitive, were given at the time of operation, but were ineffective. Autogenous vaccines administered during the postoperative period, however, controlled the organism. The patient improved rapidly, with better appetite and weight gain. In November, 1952, he neglected a severe upper respiratory infection, which led to a

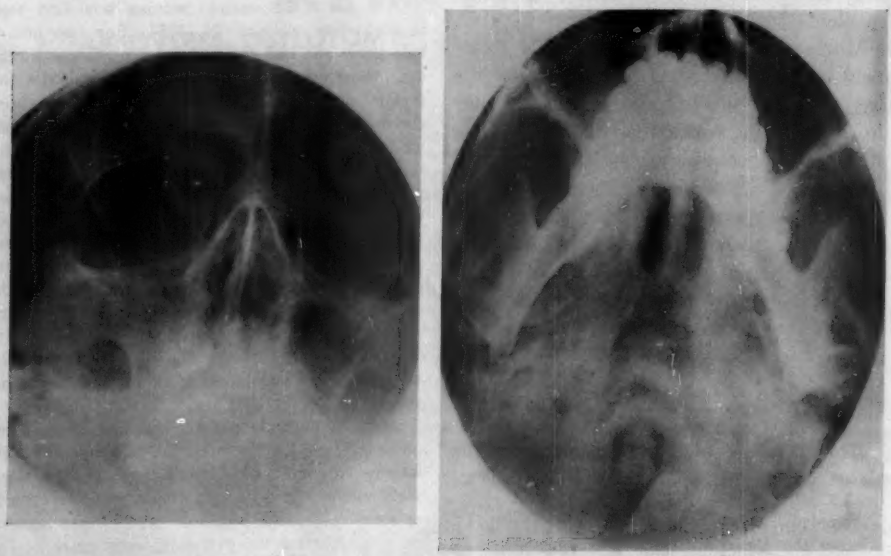

Fig. 2 (Case 3).—X-rays of sinuses showing extensive thickening of the membranes of all perinasal sinuses: maxillary, ethmoid, and frontal sinuses (left); sphenoid sinuses (right).

serious reactivation of his sinus condition. This was controlled, and since that time he has been working regularly and efficiently.

CASE 4.—A man aged 34 first came to my office on Jan. 9, 1954. For several years he had complained of arthritic symptoms of increasing severity, general malaise, and cough. In addition, for the past two months he had had eye symptoms which had been diagnosed as due to secondary glaucoma. Roentgenograms showed an extensive pansinusitis, and cultures indicated that the causative organism was a viridans Streptococcus which is sensitive to penicillin and chlortetracycline (Aureomycin). On Jan. 26, 1954, a transantral ethmoidectomy was carried out, and autogenous vaccine was given during the postoperative period. The eye condition resolved rapidly, but the arthritic symptoms responded less satisfactorily. The patient had fewer toxic symptoms than many of the others. After the operation his condition improved enough so that he was able to undertake a more active type of work than formerly.

CASE 5.—A 65-year-old man first came to my office on Oct. 20, 1953. His only complaint was severe, persistent, wheezing cough, which for the past five months had interfered with his sleep and incapacitated him for work. The referring physician had thought for a time that he had a cardiac condition. Roentgenograms of the sinuses disclosed a dense, chronic pansinusitis (Fig. 3), and cultures showed that the causative organism

Fig. 3 (Case 5).—X-ray showing extensive chronic involvement of the lining membranes of all the perinasal sinuses. The patient's only complaint was severe persistent cough.

was a nonhemolytic Streptococcus. Immunization with autogenous vaccines failed to reduce the cough. Therefore, on Jan. 2, 1954, a transantral ethmoidectomy was performed. While the patient was under anesthesia, bronchoscopy and esophagoscopy were also done, but they disclosed only tracheobronchitis. The cough slowly abated. For six months the patient has been symptom-free and has been trying to keep his excessive weight under control.

CASE 6.—A 42-year-old man first came to my office on Sept. 21, 1953. For a year he had been suffering from increasingly severe arthritis in his hands and feet, and more recently also in his left knee. These symptoms were associated with general malaise and intermittent nasal blocking. He was becoming more and more handicapped in carrying out his work. Roentgenograms showed dense, chronic thickening of the membranes in

Fig. 4 (Case 6).—X-ray showing dense, chronic thickening of the lining membranes of the right maxillary sinus.

the right maxillary sinus. Cultures disclosed a viridans Streptococcus. Some of his teeth were suspect and were removed. A week later a Caldwell-Luc operation was performed. Autogenous vaccine was administered during the postoperative period. Arthritic pain diminished strikingly in all the joints, and stiffness in some joints and hands improved considerably. Four months later the pain was entirely gone, and the patient returned to his employment overseas.

CASE 7.—An obese man aged 48 first came to my office on March 29, 1952. During the past five years he had had increasingly severe symptoms consisting of weariness, shortness of breath, wheezing cough, stiffness and pain in the joints of the lower extremities, nasal blocking and discharge. Protracted treatment of the arthritis had not been successful. He had been working only two days a week and had been able to continue working at all only because of his seniority in his union. His use of alcohol was probably not as moderate as he considered it. Beer undoubtedly contributed to his excessive weight, and whiskey increased his liability to chilling, which, in turn,

increased the frequency and severity of his sinus reactivations. Roentgenograms showed a chronic pansinusitis, and cultures revealed a viridans Streptococcus. After a combined transantral ethmoidectomy and submucous resection on April 5, 1952, the arthritic symptoms decreased rapidly. Autogenous vaccines and surgical drainage improved his condition still further, and the patient has been able to work five full days a week. If he had not been addicted to alcohol, results could unquestionably have been more satisfactory.

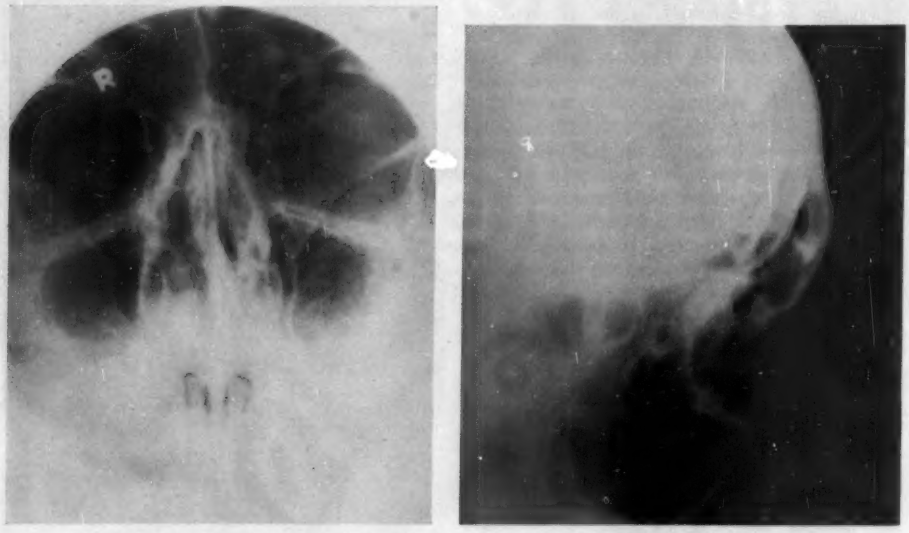

Fig. 5 (Case 9).—X-rays showing involvement of the ethmoid and sphenoid sinuses responsible for the eye condition: maxillary, ethmoid, and frontal sinuses (left); sphenoid sinuses (right).

Case 8.—A large man of 47 first came to my office on Jan. 28, 1952. For the past four years he had suffered increasingly from recurrent hoarseness, stuffiness in the left ear, anorexia, cough, pressure and pain in the chest, severe nasal blocking, and toxic symptoms. He was planning to resign his position because of increasing incapacity to do his work. Roentgenograms revealed dense, chronic thickening of the mucous membranes of all the sinuses. A hemolytic Streptococcus was cultured from the nose and throat washings. This organism is believed to be sensitive to penicillin, but a course of this drug did not relieve the symptoms. Therefore a bilateral transantral ethmoidectomy was carried out on April 15, 1952. For the past two years the patient has successfully continued his administrative duties and has even been able to indulge in his hobby, deep-sea fishing. Infrequent reactivations due to chilling or new infection have been easily controlled with penicillin.

Case 9.—A 30-year-old woman first came to my office on Feb. 25, 1952. For two years she had suffered from increasingly intense weariness, fronto-occipital headache, hoarseness, stuffiness in the ears associated with vertigo, cough, nasal blocking, postnasal discharge, and anorexia. The anorexia and vertigo were caused by Ménière's disease and were controlled by treatment of that condition. Roentgenograms of the sinuses showed that both ethmoid and sphenoid sinuses were involved but that the others were little, if at all, affected. Cultures of nose and throat washings revealed that the causative organism was a viridans Streptococcus. Since many secretory ethmoid sinus infections subside after medical treatment, autogenous vaccines were first tried, and did to some extent relieve the patient's weariness. However, early in May, 1952, she complained of a dark spot in the left eye which remained in the field of vision when she turned her eyes. The ophthalmologist diagnosed the condition as retrobulbar neuritis and recommended an operation on the sinuses. Intensive medical therapy was first tried, but without success. On July 24, 1952, a bilateral intranasal ethmosphenoid operation was done, and immunization was continued after the operation. Her eye condition subsided rapidly, and other symptoms have been sufficiently controlled to enable her to continue working.

Case 10.—A woman aged 48 first came to my office on April 20, 1949. For the past five years she had had increasingly severe malaise, generalized

muscle and joint pains, sore throat, nasal blocking, headache, and tender cervical nodes. When she was first seen her skin had a peculiar yellowish pallor. Cultures of nose and throat washings revealed the presence of a viridans Streptococcus. A course of autogenous vaccine and local treatment greatly reduced her symptoms, but on about Aug. 15, 1951, the patient noticed certain eye symptoms, which the ophthalmologist diagnosed as due to retrobulbar neuritis. Medical treatment proved unsuccessful, and roentgenograms showed evidence of chronic pansinusitis. Surgical intervention was therefore deemed necessary, and on Oct. 22, 1951, a transantral ethmoidectomy and sphenoidectomy were preformed. The eye symptoms were rapidly reduced but recurred during sinusitis reactivations whether or not there was superimposed infection. It thus seemed likely that there was some infective organism which had not been disclosed in the earlier cultures. With the use of new techniques, salivarius and S. mitis were disclosed, and in strains which are resistant to antibiotics. Immunization, with the use of controls, has kept the eye symptoms under control, and the patient's general condition has improved. A still better result could undoubtedly have been attained if the patient had refrained from excessive use of alcohol.

Case 11.—A man 48 years of age first came to my office on Oct. 17, 1952. His principal complaint was headache. Left occipital headache was always present, and left frontal headache, previously intermittent, was becoming persistent. He also complained of nasal blocking, postnasal discharge, voice weakness, malaise, insomnia, and generalized neuralgia from time to time. His history included a "nervous breakdown" during the preceding February and injury to the cervical spine 20 years before. Neurologic examination gave no useful information. In addition to a fractured nasal septum, the patient had the darkish-red inflammation of the pharyngeal mucosa which is characteristic of chronic disease. Roentgenograms taken on Oct. 25, 1952, revealed pansinusitis involving predominately the left perinasal sinuses. Cultures from nose and throat washings disclosed hemolytic Staphylococcus aureus and nonhemolytic streptococci. An autogenous vaccine was administered before operation, and a bilateral transantral ethmoidectomy was done on Nov. 13, 1952. The patient had no headache during the postoperative period, but it recurred about a month later. Very large doses of penicillin were used during, and occasionally after, the postoperative period in an effort to prevent reactivations, which are characteristic of this condition. Reculture in April, 1953, revealed a strain of viridans Streptococcus which is resistant to all antibiotics. Since the headaches continued and the mucosa still showed the coloring characteristic of disease, vaccines made from the resistant organisms were injected, but with unsatisfactory results. It became increasingly evident that the patient was a moderate but continuous user of whiskey, and he could not be persuaded that alcohol aggravated his disease.

Case 12.—A man aged 46 first came to my office on Aug. 3, 1954. For 12 years he had suffered from recurrent nasal blocking, postnasal discharge, general malaise, and neuralgia—all of which had become persistent during the past year. He described himself as a moderate drinker. The nasal and pharyngeal mucosa showed the coloring characteristic of chronic disease, and cultures from the nose and throat disclosed a viridans Streptococcus and a nonhemolytic Streptococcus as the causative organisms. Roentgenograms disclosed an advanced chronic pansinusitis. Accordingly, transantral ethmoidectomy and submucous resection were performed on Aug. 20, 1954. Pathologic examinations confirmed the diagnosis of chronic bilateral sinusitis. The patient responded well to administration of autogenous vaccines during the postoperative period, but the darkish-red color of the mucosa persisted. In April, 1955, recultures revealed the original organisms were still present. In spite of urging on my part that he stop drinking, the patient has persisted in using alcoholic beverages.

Case 13.—A man 45 years old was first seen in my office on March 23, 1952. He complained of nasal blocking, postnasal discharge, cough, burning sensations on swallowing, frequently severe malaise, and intermittent pain in the right shoulder, which varied with the malaise. Eight years earlier he had been in the hospital for 10 days because of his condition. Roentgenograms showed an advanced chronic pansinusitis involving the ethmoid and maxillary sinuses. Cultures from the nose and throat washings disclosed hemolytic S. aureus and a viridans Streptococcus. Autogenous vaccines and large doses of penicillin were given during periods of activation, which were frequent and protracted, but the patient responded only very slightly to this treatment. In January, 1954, the patient was admitted to the hospital because of an extremely severe activation of all symptoms, and a bilateral transantral ethmoidectomy was done on Jan. 21. The pathologist reported chronic inflammation. For some time after the operation the patient's symptoms were under better control, but during the fall of 1954 tended to recur in aggravated form. Recultures showed the presence of nonhemolytic streptococci. In spite of immunization, the dark-red color indicated that chronic disease persisted. Finally, however, after his complete abstinence from alcoholic drinks, there was definite improvement.

Case 14.—A man aged 63 was first seen in the hospital in May, 1954, suffering from severe malaise, persistent wheezing cough, nasal block-

ing, postnasal discharge, and tender cervical nodes. Roentgenograms showed an extensive pansinusitis, and cultures from nose and throat washings disclosed nonhemolytic staphylococci and a viridans Streptococcus. On May 18, 1954, submucous resection and transantral ethmoidectomy were done. During the postoperative period immunization procedures were used. After the operation the patient's condition was considerably better, but in the fall of 1954 he suffered a severe reactivation of the sinusitis associated with difficulty in swallowing. His response to antibiotics was poor, and nasal and pharyngeal color indicated chronic disease. Fluoroscopic and roentgen studies of the esophagus gave no information, nor did a direct esophagoscopy done in November, 1954. However, the patient has had fewer activations of sinusitis since the operation, and as he has stopped completely using alcoholic drinks his condition has gradually improved.

Causes

Williams[2] has defined allergy as "an acquired alteration in the capacity to react, which is predicated on an antigen-antibody mechanism." Allergic responses fall into three categories: anaphylactic, eczematous, and bacterial. It is to the latter group that the chronic perinasal sinus conditions under discussion belong. In these cases, the organisms act as the allergens and behave like pollens, except that they are endogenous. The proteins of the organisms are the primary factors inciting the allergic response.

Clinical investigations on the subject reveal that fully 75% of sinus infections are caused by staphylococci, most frequently by S. aureus. During the acute stage staphylococci are rarely resistant to adequate dosage of antibiotics, and the subacute stages are also to a large extent controllable by such medication. In either stage, surgical procedures may also be necessary, and staphylococcic toxoid is of great value in controlling allergic and inflammatory symptoms.

Of the causative organisms involved in the other 27% of cases, most are also Gram-positive. Bacillus proteus and pyocyaneus occasionally occur; but by far the most important are the streptococci, of which viridans Streptococcus and S. mitis (Pneumococcus Type 3) are especially prominent. This is largely due to the fact that many strains of these organisms are totally resistant to any antibiotic presently in use. The hemolytic Streptococcus is more easily controlled than most strains of streptococci because it is sensitive to antibiotics. Contamination by fungi is becoming more frequent because of the necessity of using large doses of antibiotics over long periods of time.

Pathology

The bones are not affected by bacterial allergy, though a fractured septum or a cystic middle turbinate may cause partial blocking. The basic pathologic change is a swelling or thickening of the mucosa which lies over the bone in the sinuses. As the case matures over the course of 6 to 15 years, this swelling or thickening becomes more extensive and increasingly severe, until it reaches a point where it can no longer be reversed by medical treatment alone.

The blood supply to the nose and perinasal sinuses is normally abundant. However, in the presence of swelling or thickening due to chronic or acute infection, the normal blood supply is decreased. Moreover, during activations the blood supply diminishes relative to the lymphatic drainage from the sinus areas. Thus there exists an increased gradient of toxic and allergic material coming into relation with the antibodies produced in the sinus membranes and those available from the restricted blood supply. Consequently, greater amounts of toxic and allergenic material enter the general circulation through lymphatic drainage from the sinus area.

Symptoms

The condition is remarkable for the variety of symptoms from case to case and for the ability to simulate or complicate other diseases. Local symptoms consist primarily of nasal blocking, sneezing, watery and persistent discharge, postnasal discharge, dryness of throat and nasal pas-

sages, and, reactivated cases, actual pain in these areas. Most cases also have general toxic absorption symptoms, especially malaise, weakness, depression, anorexia, generalized neuralgia, and insomnia. Some few patients have no local symptoms at all.[3]

Associated with the primary and more usual symptoms, there may also be tightness or dullness between or around the eyes, tenderness or pain in the back of the neck and occiput, extending at times into the shoulders. Eustachian catarrh may occur, leading to stuffiness in the ears or associated with impaired hearing and vertigo. Other patients may complain of tenderness and swelling of the cervical nodes or tenderness over the sinus areas.

Asthma is a possible complication, but allergic wheezing due to tracheobronchitis and persistent cough without asthma are commoner. The cough is typically brassy, and may be productive, especially in the morning. The voice may be weak or actually hoarse. Some patients have a sense of fullness in the throat or even a sense of swallowing over a lump. Others have a burning sensation on swallowing due to associated esophagitis. Headache is frequently spoken of as a prominent symptom. Actually it does not usually occur unless there is an acute infection superimposed on the chronic condition.[4] Associated gastritis often causes nausea and heartburn, and with these there may be enteritis, flatulence, and distension. The patient swallows irritating discharge continually 24 hours a day, which can be a significant cause of such disturbances.

Course of the Disease

The course of the disease is most commonly characterized by a pronounced tendency to cyclic reactivation. At first the condition may be so slight that only occasional activations are noted by the patient. Episodes may occur at intervals of from a few weeks to a few months. They are usually precipitated by events which would hardly be noticed by a healthy person—chilling, fatigue, or both. Symptoms which the person exhibits during quiet phases are enormously augmented during episodes of activation, and as the disease progresses, reactivations become severer and more protracted. Thus the patient is in a continual state of imbalance, varying between periods during which the sinus is relatively quiescent and he is merely generally ailing and acute phases when he is exceedingly ill.

Successful treatment depends, then, partly on the patient's fully understanding the importance of protecting himself from situations which may initiate reactivations. If he is careless about drafts, forgets to carry an extra light wrap in case of unexpected changes in the weather, or increases the danger of chilling by drinking alcoholic beverages, he will probably be subject to frequent and perhaps severe reactivations. He must also be careful about exposing himself to infections, and if such exposure does inadvertently occur, he must take all precautionary measures, including, of course, seeking medical assistance. Without intelligent cooperation from the patient, the most skilled medical or surgical treatment will be at least partially defeated. It is sometimes thought that changes in climate benefit these patients. Actually, this is not true and should not be advised.

Another problem in the management of the disease and of its reactivations is one which occurs in relation to other chronic diseases as well; that is, the patient's consumption of alcoholic beverages. The use of alcohol produces a distinct reaction or engorgement of the mucous membranes, which is an additional irritant to the already diseased mucous membranes of the nasal sinuses. The engorgement from this source is in addition to the augmented inflammation from activation due to chilling, overfatigue, or both. In a society where the expense-account system permits opportunity for more than usual indulgence in alcoholic drinks, some sort of such drinking has become a widespread routine. Many of my patients have reported to me that in a

matter of minutes after taking a drink local symptoms of the sinus disease are distinctly augmented. The engorgement of the mucosa resembles that of the laryngeal mucosa. It is already well recognized that under such circumstances the use of alcohol is to be forbidden. This does not refer to the alcoholic, but to the casual drinker with chronic sinusitis, whose disease may be considerably aggravated and may be more difficult to control with only moderate drinking. The reviewed Cases 7, 12, 13, and 14 illustrate this problem.

Medical Treatment

Many cases of sinusitis can, and should be, treated by medical means. When indicated, the assistance of the internist, the ophthalmologist, or the allergist may be essential. Dust extracts, Staphylococcus toxoid, or autogenous vaccines are useful in early cases. Certain of the common contributing allergies should be checked, such as histamine, dust, Staphylococcus toxoid, spring grasses, and rag weed. The tying-in of these allergies with the basic bacterial factor may render the case uncontrollable. Desensitization can be carried out along with immunization. Moreover, the anti-allergic benefits of autogenous vaccines are more pronounced when intradermal injections are given. Oral antigenic material, which I have used more recently frequently, gives strikingly beneficial results in those cases where the injectable autogenous vaccine is unproductive. A combination of these two types of vaccine therapy is difficult, but rewarding; great care must be exercised to keep below the reaction level. Patients under cortisone therapy are seldom benefited, since there is very poor antibody production with immunization in the presence of this drug.

These measures are, however, rarely useful in treating mature cases except during the postoperative period, when they should be employed. Antibiotics may be helpful. The first 15 of the present series of 65 cases were treated without immunization or desensitization. In the next group, of 30 cases, all received autogenous vaccine and, when indicated, Staphylococcus toxoid.[5] Symptoms were better controlled in this group than in the first. The last 20 cases have been given autogenous vaccines. As a control, titration tests were made from the morning urine for toxic products.[6] While space does not permit discussion of all these cases, symptoms were much better managed in this last group than in either of the others. This method is particularly promising in treating patients who have symptoms of arthritis as well as chronic bacterial allergy.[7]

Surgical Treatment

However, medical treatment cannot provide drainage of blocked sinuses, nor can it remove the hopelessly diseased, thickened mucosa which lines the perinasal sinuses in mature cases. For these, surgical measures are necessary. Operations developed by Lore[8] and Luongo[9] and by others make it possible today to operate on the perinasal sinuses without destroying functional nasal structures, and thus it is possible to avoid the postoperative atrophic rhinitis which was so common in earlier times. From the available techniques and combined procedures now available, a method can be found which will prove successful if the special requirements of the individual patient are considered.

The submucous resection of the septum which divides the two sides of the nose is the basis of all nasal surgery. This operation not only removes blocks due to fracture or dislocation of the septum, if present, but also makes infraction of the middle turbinates possible. The turbinates must be preserved; they are the functional tissues of the nose acting to condition the inspired air which will eventually enter the lungs. Procedures which include removal of the turbinates lead inevitably to the dry, crusty tissues of postoperative rhinitis. The patient is left with a nose that is chronically unhealthy and never functions normally.

With modern procedures, such an eventuality is unnecessary, and indeed unforgivable. Infraction of the turbinates makes it possible to remove polyps, if present, without destroying tissues. If any of the turbinates themselves show polypoid degeneration, they can be stripped, allowing normal mucosal regeneration. If there is a cystic condition in the turbinates, the ethmoid cells within them can be crushed, thus eliminating the block without destroying the functional tissues. Infraction of the turbinates also facilitates postoperative care of the intranasal ethmoidectomy cases. This operation is particularly useful in treating patients who have eye complaints secondary to perinasal sinusitis.

Of particular interest are the pansinusitis cases, in which all perinasal sinuses are involved. The treatment preferred for such cases is bilateral transantral ethmoidectomy. This procedure combines a radical maxillary sinus operation (Caldwell-Luc) with ethmoidectomy, which is done through the maxillary sinuses. The advantages of this approach are that obviously no functional tissue is lost, since the nose is not entered,[11] and that the ethmoidectomy is easily carried out because the area is readily reached through the maxillary sinuses. My own belief is that in these cases submucous resection, infraction of the middle turbinates, and intranasal ethmoidectomy should be combined with transantral ethmoidectomy. This combination greatly facilitates drainage from the infected area, simplifies the approach to the sphenoid sinus in case it needs operation or irrigation, and makes it possible to give postoperative care to the affected parts, especially in the ethmoid region. Above all, leaving the turbinates in place preserves the functional turbinate tissues and ensures a healthy nose free from atrophic rhinitis. When this combination is properly carried out, it is rarely necessary to resort to external operations on the frontal sinuses unless there are severe complications, such as brain abscess, meningitis, cavernous sinus thrombosis, or osteomyelitis of the skull.

Summary

Sixty-five cases of chronic bacterial allergy of the perinasal sinuses are reviewed. The type of organism is important, since the allergen is the organism which caused the infection.

Staphylococci cause 75% of sinus infections. Most of the resistant organisms found in this series were streptococci.

Symptomatology is varied and may simulate many other diseases.

Since the disease is easily reactivated by chilling, fatigue, and reinfection, patient cooperation is essential to successful treatment. Changes in climate are not beneficial and should not be advised.

Early cases are usually controlled by medical measures, including the newer forms of desensitization and immunization with controls. The use of such measures is clearly indicated in all cases.

Mature, or very advanced, cases can seldom be controlled by medical measures alone.

Radical surgical procedures should be carried out in advanced cases without destruction of functional tissues.

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Direct Adenoidectomy

II. Technical Aspects

PAUL GUGGENHEIM, M.D., Topeka, Kan.

Numerous techniques have been described both for exposing the nasopharynx to direct view and for dealing with the diseased adenoid visualized. A historical resumé of these was presented in two previous publications.* The purpose of the present paper is to discuss these methods in a comparative way. This will be done under several headings.

Shortcomings of Blind Adenoidectomy

Although nowhere explicitly stated as such, a "decompressive" concept of adenoidectomy seems to have been implicit in much of the thinking about adenoid surgery before the introduction of the direct technique by Andrew Love [3] and L. K. Guggenheim.† While even a poorly done adenoidectomy will usually relieve postnasal obstruction due to a large central adenoid mass, such incomplete surgery will frequently fail to prevent recurring bouts of otitis media or to alleviate conduction hearing impairment due to obstruction of the Eustachian orifices. We know that many children whose nasopharynges are literally stuffed with huge masses of adenoid tissue escape the ravages of otitis media, while others, harboring strategically placed adenoid masses of much smaller size, suffer when these masses are the seat of infection and edema. The tissue responsible for this phenomenon frequently consists of the paired lateral lymphoid curtains. These structures are a part of Waldeyer's ring and commonly associated with the adenoid, although they are really quite separate from the central adenoid mass. They were shown by L. K. Guggenheim‡ to lie in intimate contact with the Eustachian orifices and to be incapable of resection by means of adenotomes, curets, or other means commonly employed.

While the lateral lymphoid curtains must by no means be resected in all adenoidectomies, the vault should always be cleaned of central adenoid. Many operators feel that they can detect remnants with the index finger. The surgeon who wears rubber gloves will have perhaps less confidence in his ability to do this than one who operates barehanded. In any event, remnants detectable by such palpation are larger than those under discussion.

Lighting

It is universally agreed that strong direct illumination from an electric headlight gives the best result. The standard ceiling lamps of the operating room are inadequate for illuminating the nasopharyngeal vault, and no adjustable floor lamp will serve without its rays being blocked by the manipulations of the surgeon. L. K. Guggenheim used a reflecting headmirror, sterilized with benzalkonium (Zephiran) and manipulated by a sterile clothespin. A 100 watt bulb was placed just to the left of the patient's head. I have operated with this method and find its only disadvantage to consist in the threat of the hot bulb to the anesthetist's bare arms. Of the various electric headlights available, the best is the Cameron light, which consists of a powerful naked bulb mounted before a small parabolic mirror. This gives vastly better illumination than the type of apparatus in which the bulb is housed in a black cylindrical shell with an angled plane reflector at its lower end.

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Article illustrated by the author.

* References 1 and 2.

† References 4 and 5.

‡ References 4 and 5.

Anesthesia

Ideally it is desirable for general anesthesia to be deep enough to abolish pharyngeal reflexes, permitting full relaxation of the soft palate. With very young children, in whom deep anesthesia is dangerous, experience will enable the operator to obtain adequate exposure of the nasopharynx without it. By gradual, gentle traction on the soft palate with the Love retractor, a certain degree of palatal spasticity may be overcome and exposure maintained with little difficulty.

Endotracheal intubation has been advocated by Meltzer § as a routine procedure. This would certainly be mandatory if the operation were to be carried out, as Meltzer prefers, with the patient in the sitting position. With a supine patient it would appear to be an optional matter. My own feeling is that routine intubation, except in the hands of highly skilled anesthesiologists or nurse-anesthetists (who are not always available outside large medical centers), would probably lead to more laryngeal complications than it would avoid pulmonary ones. Trendelenburg position and adequate hemostasis should suffice to protect the tracheobronchial tree.

In adults, general anesthesia is not always necessary for adenoidectomy but is certainly justified in case of doubt. If general anesthesia is to be employed, anesthetists prefer to intubate all cases. If local anesthesia is contemplated, the patient should be well premedicated. No mouth gag is used. If tonsillectomy is to be done, this is carried out first in the semisitting position so that the patient may relax, yet easily turn his head to expectorate. For the ensuing adenoidectomy he is placed in mild Trendelenburg position. The nasopharynx is anesthetized in the following manner. A spray of 2% tetracaine (Pontocaine) or 10% cocaine is used sparingly. After shrinking the nasal mucosa with 1% ephedrine or phenylephrine (Neo-Synephrine), a cotton stick applicator moistened in the topical anesthetic solution is inserted along the floor of the nose on either side into the nasopharynx and left for at least three minutes. This maneuver establishes a good anesthesia of the nasopharyngeal surface of the soft palate, permitting greater relaxation. After removal of the applicators, a preliminary direct inspection is carried out and a gauze sponge moistened in the topical anesthetic solution applied for a minute or so to the vault. After this, procaine, 1%, with epinephrine hydrochloride, 1:1000 (eight drops to the ounce), is injected into the submucosa with a tonsil needle, through the adenoid mass on either side. A small amount may be injected just above Passavant's band if central adenoid tissue extends this low, a rarity in the adult. An injection higher in the vault is necessary in any case. The lateral curtains are separately injected, if present, usually in two places on either side. After this, it is possible by gradual, gentle retraction to gain the confidence of the patient's soft palate and he will relax it. Occasional reflex swallowing causes no difficulty. Access to the remotest recesses of the nasopharynx is just as readily obtained as in a child under ether anesthesia.

Position

Andrew Love, L. K. Guggenheim, and their followers did not find it necessary to abandon the supine posture of the patient in order to carry out direct adenoidectomy. Meltzer ‖ and others on the Eastern Seaboard have, for years, operated upon children in the sitting posture. It would seem to be a matter of election which posture to adopt. Theoretically, it should be possible to translate any spatial relation between surgeon and patient through a 90 degree rotation to reproduce the same degree of access to the nasopharyngeal space.

The Rose position has always seemed to me to place the surgeon at a disadvantage in performing adenoidectomy, since he is facing the hypopharynx rather than the epipharynx. I prefer the conventional posi-

§ References 6 and 7.

‖ References 6 and 7.

tion, opposite the patient's middle on the right side.

Self-retaining tongue depressors, particularly the Crowe-Davis gag, are commonly employed when the Rose position is adopted. I have always been inclined to agree with Meltzer that such apparatus was to be avoided because, with the tongue fixed, inadequate mobility of the palate was attainable for proper access to the nasopharyngeal vault. I am bound to admit, however, that I have recently observed excellent exposure and results with this method in the hands of G. O. Proud and his staff at the Kansas University Medical Center. The tori and fossae of Rosenmüller are quite adequately exposed. However, Proud finds it frequently necessary to use a nasopharyngeal mirror in order to visualize the upper reaches of the vault, in particular the choanae and septal root.

Preliminary Operative Procedure

If tonsillectomy is contemplated as part of the procedure it should be carried out first. The soft palate and, especially, the posterior tonsil pillars are then easier to retract, permitting better exposure of the whole nasopharyngeal space, particularly its lateral recesses.

After completion of tonsillectomy, gentle elevation of the soft palate will give an estimate of the size of the adenoid mass. In the adult in whom any central adenoid is likely to be confined to the higher regions of the vault, somewhat more extensive retraction of the palate is necessary for this preliminary inspection.

Following the preliminary direct inspection, the palate retractor is laid aside. The La Force adenotome is then employed blindly. One uses the largest instrument which will fit into the cavity. The tonsil suction, held in the operator's left hand, will serve both to depress the tongue and to keep the field reasonably dry during the few moments required for three to five bites with the La Force instrument.

Attention is next directed to hemostasis. The brisk bleeding which invariably accompanies use of the La Force adenotome is terrifying only to the uninitiated and should never be used as an excuse for omitting further steps in the procedure. Gauze sponges sparingly moistened in epinephrine hydrochloride, 1:1000, should be introduced on sponge forceps and gentle pressure maintained for a period equal to or exceeding the bleeding time—usually three to five minutes. As a rule, even the nasopharynx of a small child will admit two such sponges. In adults, a third sponge may sometimes be advantageously introduced. Since the use of a third sponge forceps is awkard, it is more convenient to hold the additional sponge in a curved hemostat. Steady pressure throughout the cavity is achieved in this way and is far superior to blotting rapidly about with a single sponge. Tannic-acid powder should be avoided at this stage because it tends to obscure the difference in texture between lymphoid tissue and clean nasopharyngeal submucosa.

It is here, unfortunately, that all too many adenoidectomies end. Precisely at this point the otolaryngologist's real challenge begins.

A thorough direct inspection of the nasopharynx is now called for. With proper armamentarium and technique, the upper reaches of the vault may be exposed in all cases irrespective of the shape of the nasopharyngeal cavity. Reeves and Brill [8] have described the classification of possible configurations according to the French anatomist Testut, and Meltzer [9] refers to the variations in shape depending on cranial index, first described in 1901 by Escat and by Moure and Lafarelle (quoted from Dickson and McGibbon [10]). Far more significant than any possible difficulty which these anatomic variations may cause in performing direct adenoidectomy (I have never been aware of any) are the uncertainty of placement of the nasopharyngeal radium applicator, pointed out by Dickson and McGibbon,[10] and the unsureness of blind adenoidectomy, stressed by Guggenheim ¶ and Meltzer.[9]

¶ References 4 and 5.

In common with many others, I have found the Love soft palate retractor an ideal instrument for obtaining direct exposure of all parts of the nasopharynx. The instrument should be held in the surgeon's left hand, handle crossing the upturned palm at its juncture with the bases of the fingers, the hook being draped over the base of the index finger. In action, the hand tends to assume a somewhat vertical posture, the ulnar side being directed toward the floor as well as toward the patient's feet (Fig. 1).

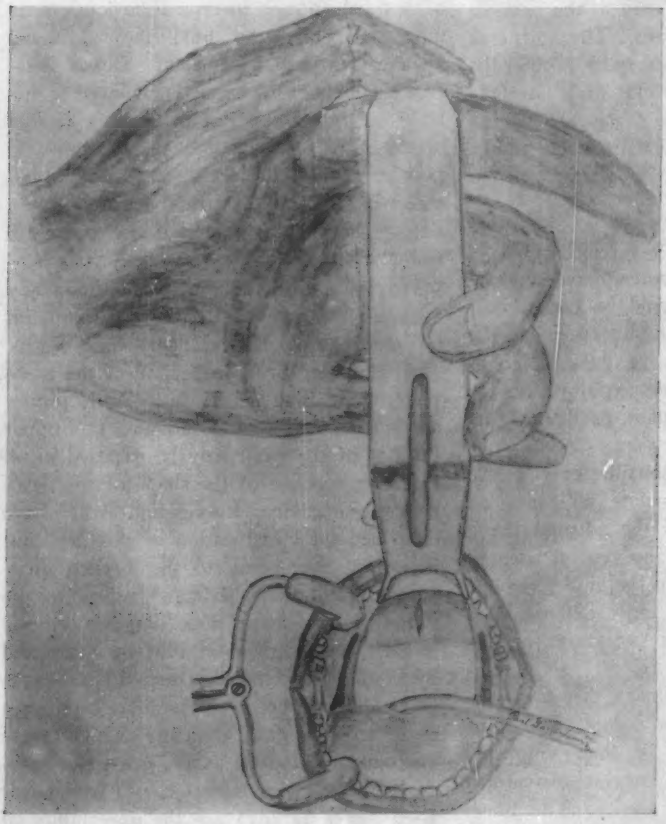

Fig. 1.—Basic direct view of clean nasopharyngeal vault with Love retractor held in cephalocaudal (sagittal) axis.

This view is bounded anterosuperiorly by the retractor itself, which engages the free edge and only a couple of millimeters of the soft palate. The eye should be able to pass from the retractor tangentially along the nasopharyngeal surface of the soft palate into the vault, where the septal root may be identified. The view is framed laterally by the posterior tonsil pillars, which are put on stretch. Inferiorly, the view is bounded by the tongue and, posteriorly, by the oropharyngeal wall, which, in the absence of the central adenoid mass, sweeps upward to the exposed nasopharyngeal vault with a quite gentle slope. The lateral recesses of the nasopharynx, including the lateral lymphoid curtains, tori tubarii, and fossae of Rosenmüller, are mostly hidden by the posterior pillars. When their exposure is necessary, the palate retractor is slid obliquely behind the posterior pillar of the side desired so that it assumes an angle of approximately 45 degrees with the sagittal plane (Figs. 3, 4, 5, and 6). It will be noted

that the Love retractor is wide enough to extend across the entire width of the soft palate—from one posterior pillar to the other. It thus permits retraction of the soft palate en masse. Narrow, ribbon-like retractors, such as the Jackson, Goodyear, etc., permit only a tenting of the velum, which represents but a slight improvement over what one can accomplish with a Hurd dissector.

Cleaning Up the Vault

With the vault directly exposed, any remnants of the central adenoid mass are removed with L. K. Guggenheim's [4] special small La Force adenotome # (Fig. 2*e*), which has an inside-basket width of only 9 mm. Occasionally some tissue near the septal root cannot be engaged with this instrument, but is easily grasped and avulsed with small triangular holding forceps (Fig. 2*d*) or a curved Allis clamp, or bitten away with a punch forceps or ethmoid biting forceps. During this clean-up, the tonsil suction may be either delegated to the nurse-assistant or retained in the operator's right hand along with the delicate small adenotome.

I have never used a curet but believe, like Meltzer,[7] that this instrument is safe if used gently under direct vision. Used roughly or blindly, a curet can lop off pieces of tubal cartilage or remove the full thickness of nasopharyngeal submucosa with resulting scarring.

The attempted abrasion of adenoid remnants with the gauze-covered finger has been rightly condemned as useless by L. K. Guggenheim [5] and Meltzer.[7]

The hemostatic procedures mentioned previously are repeated after the vault is cleaned. Any persistent bleeding points are

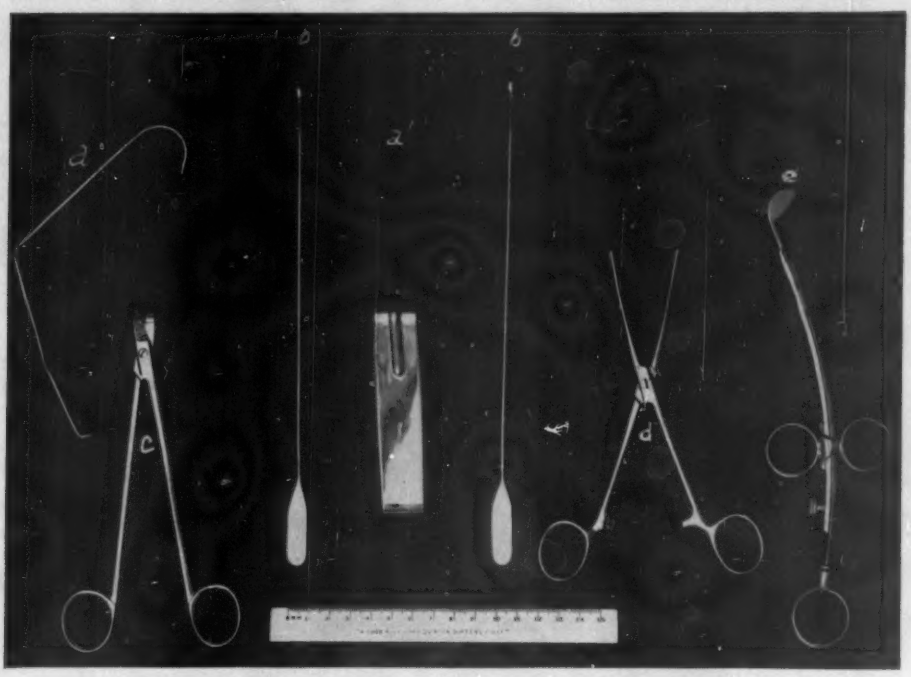

Fig. 2.—Special instruments for direct adenoidectomy: (*a,a'*) Love soft palate retractors; (*b,b'*) Gyergyai olives, right and left; (*c*) Guggenheim's small, right-angle curved scissors; (*d*) triangular holding forceps; (*e*) Guggenheim's specially small adenotome.

This instrument is manufactured by Storz and by V. Mueller.

seized for three to five minutes with Jackson duck-billed hemostats. If necessary, slip-knot ligatures of No. 000 plain absorbable surgical (gut) suture strands may be tied over the Jackson clamps, as is commonly done in the tonsil fossae. Since this maneuver calls for both the operator's hands, the Love retractor is delegated to the assistant, who must be trained to maintain it in correct position. Occasionally it is desirable to pass a transfixion suture across the vault to approximate the edges of the median raphé, which are sometimes a source of persistent bleeding. For this purpose I have found an ordinary diamond-pointed slip-proof needle holder better than any of the automatic suture passers. If no further surgery is indicated, tannic-acid powder on an epinephrine-moistened sponge may be used to frost the exposed submucosal surface. Some prefer thrombin solution.

fer an instrument of this type. Quite satisfactory ablation of the curtains can be carried out from the lower end of the posterior aspect of the tonsil pillar all the way up over the convexity of the torus by this means. For the lymphoid tissue of the fossa of Rosenmüller, however, a finer-pointed instrument is required, e. g., an ethmoid biting forceps.

Any punch forceps has the disadvantage of grasping and biting in a single operation—a feature which makes depth of excision hard to gauge, since the tissue is not on stretch. Repeated biting away at it is necessary. In an effort to overcome this defect, L. K. Guggenheim [4] invented a lateral curtain punch somewhat resembling a small vise. The jaws of this instrument were slipped over the lateral curtain after the latter had already been seized with his small triangular holding forceps and put on

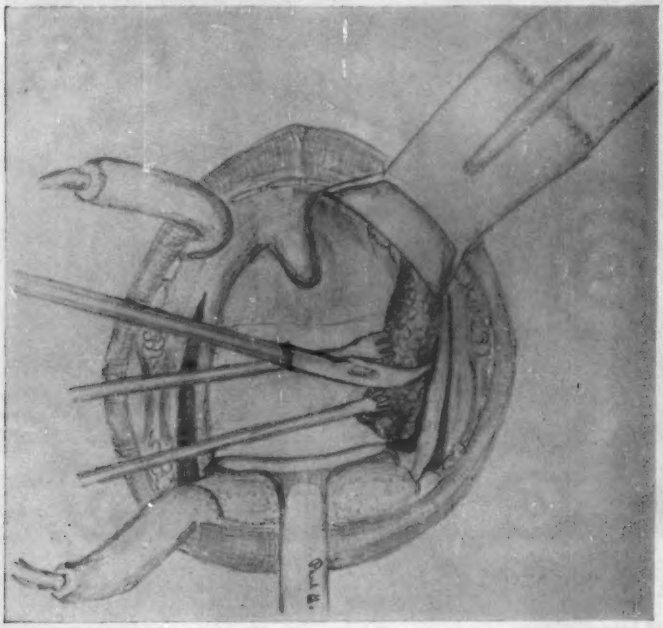

Fig. 3.—Exposure of left lateral curtain by oblique application of Love retractor. Preliminary hockey-stick incision to delineate lateral curtain in relation to posterior tonsil pillar.

Resection of the Lateral Lymphoid Curtains

Love [3] published a description of a punch forceps for biting out the lateral adenoid tissue piecemeal. Most operators today pre-

stretch. This he later * abandoned in favor of a method of sharp dissection by means of a specially devised small, right-angle curved scissors. To this method one refine-

* References 4 and 5.

ment has since been added which perhaps justifies describing it anew. The procedure is admittedly somewhat more difficult to learn than use of punch forceps; but I find it more anatomical and, having encountered anomalous internal carotid arteries in the submucosa beneath the curtain in two children, safer.

The right lateral curtain is first exposed (Fig. 3) by oblique application of the Love retractor as described previously. In a very small percentage of cases in which the curtain is very large or in which the posterior tonsil pillar is less well developed than usual, the lip of the Love retractor may show a tendency to slide from behind the pillar into the tonsil fossa, obscuring the operative field. If this difficulty is encountered, one may gently retract the posterior pillar laterad and slightly cephalad with the Hurd dissector. This suffices in the early stages of the dissection, since one begins this far below, at the base of the tonsil pillar. After gentle retraction of the pillar, the curtain is seized with two long-handled Allis forceps,† which are then handed to the assistant, who exerts a pull anteromedially.

At this point, L. K. Guggenheim ‡ engaged the inferior extremity of the curtain by straddling it with the blades of his specially constructed small, right-angle curved scissors (Fig. 2*c*). Having several times inadvertently fenestrated the posterior pillar during the upward dissection of the curtain from it by too-deep penetration of the scissors, I have introduced a preliminary mucosal incision which delineates the curtain from the pharyngeal attachment of the posterior pillar on the lateral side and from the base of the pillar inferiorly (Fig. 3). A Bard-Parker No. 12 blade (crescentic) on a long-handled knife is used for this purpose. A short, horizontal incision is first made to free the lower end of the curtain, not more than 2-4 mm. in length. From the lateral end, the incision is carried perpendicularly up to the torus, or as near it as possible. The result is a "hockey-stick" wound which permits the Allis clamps to mobilize the curtain much more satisfactorily. The bulk of the attachment to be severed is somewhat reduced, and the bed of the dissection is defined so that injury to the pillar is less likely to occur.

My two cases of anomalous internal carotid arteries were encountered since adopting this incision. In both instances, strong otologic indications demanded resection of the curtain, which, in each case, was enormously and asymmetrically hypertrophied on the side of the anomalous vessel. The preliminary incision at once revealed the pulsating vessel just beneath the submucosa. The ensuing dissection was carried out with ease and confidence.§

Excision is carried upward to the level of the torus (Fig. 4). Although the curtain is anatomically continuous with the lymphoid tissue overlying the torus and filling the fossa of Rosenmüller, it is best to amputate

† This method is preferable to use of L. K. Guggenheim's [4] triangular holding forceps. This instrument is very useful in removing remnants of the central adenoid mass high in the vault, but it has been found to slip off the lateral curtain due to the fact that it has only gentle corrugations parallel to the length of the base of the triangle. Teeth biting into the curtain are needed, but the instrument has never been so modified.

‡ References 4 and 5.

§ Otologic results were excellent in both cases. Yet some might hesitate to risk injuring or at least exposing the artery in performing such a dissection. Since the adenoid usually atrophies in adolescence, the anomalous vessels would be destined, in any event, to lie in the submucosa, unprotected by any lymphoid tissue. Such a case was actually observed in 1952. The patient, a man of 55, complained of a right otalgia of three months' duration. Examination disclosed a fusiform aneurism, perhaps slightly greater than a centimeter in diameter, of an anomalous right internal carotid artery projecting into the lumen of the nasopharynx. The patient was referred to the Mayo Clinic, where the neurosurgical consultant considered the risk of rupture of the aneurism far less than that of ligation of the artery. During the period of the patient's complaint he was under severe psychic stress due to a tragic home situation. When this stress was somewhat alleviated, the otalgia disappeared. He is still healthy.

the superior pedicle of the curtain at the level of the anterior end of the torus. The Love retractor is essential here. Even if, as rarely happens, a poorly developed posterior pillar has made one resort to the Hurd for retracting the pillar during resection of the lower part of the curtain, removal of the lower segment will deepen the gutter behind the pillar so that the Love retractor can be employed without slippage for resection of the upper segment. The superior pedicle of the curtain is most conveniently severed with ordinary tonsil scissors (Fig. 5). Not infrequently, prior careful dissection with the small, right-angled curved scissors will have so attenuated this pedicle that it is easier simply to avulse it.

Lymphoid tissue on the convexity of the torus may then be bitten off by a punch forceps or seized with a long-handled Allis forceps and dissected off with the small, right-angled scissors. Simple avulsion by the clamp sometimes suffices. For evacuating the fossa of Rosenmüller I have been in the habit of avulsing the lymphoid tissue piecemeal with the Allis forceps. The loose, somewhat areolar structure of the tissue facilitates this, as does the fact that the Allis clamp has approximately the same width as the fossa itself. Lately an ethmoid biting forceps has been used with very satisfactory results.

After resection of the right curtain is completed, bleeding is readily controlled by application of a single epinephrine-moistened gauze sponge to the bed of the curtain for three minutes.

The procedure is repeated on the left side.

Removal of the lateral curtains further accentuates the impression conveyed by the direct view that the nasopharynx is much broader than one conceives it from the mirror view. The unbroken sweep from

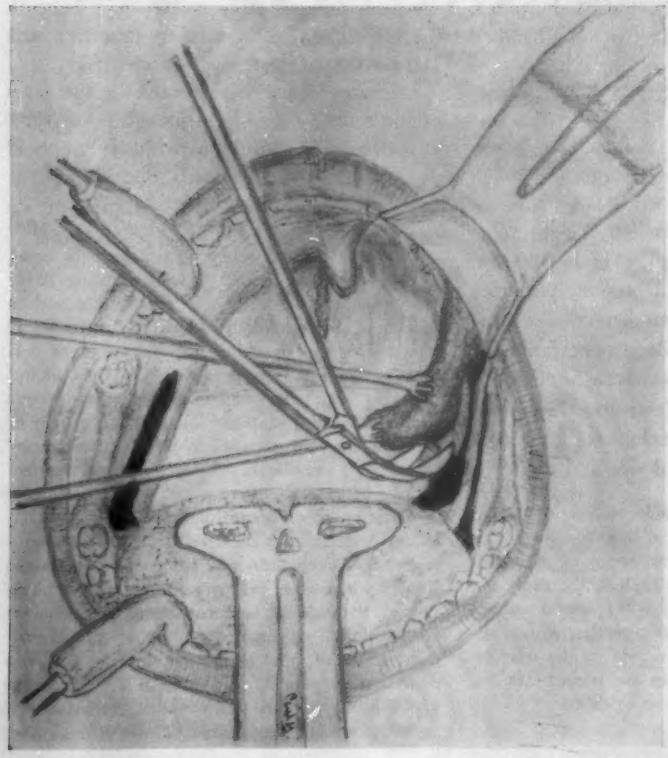

Fig. 4.—Dissection of the lateral curtain with the Guggenheim small, right-angled scissors.

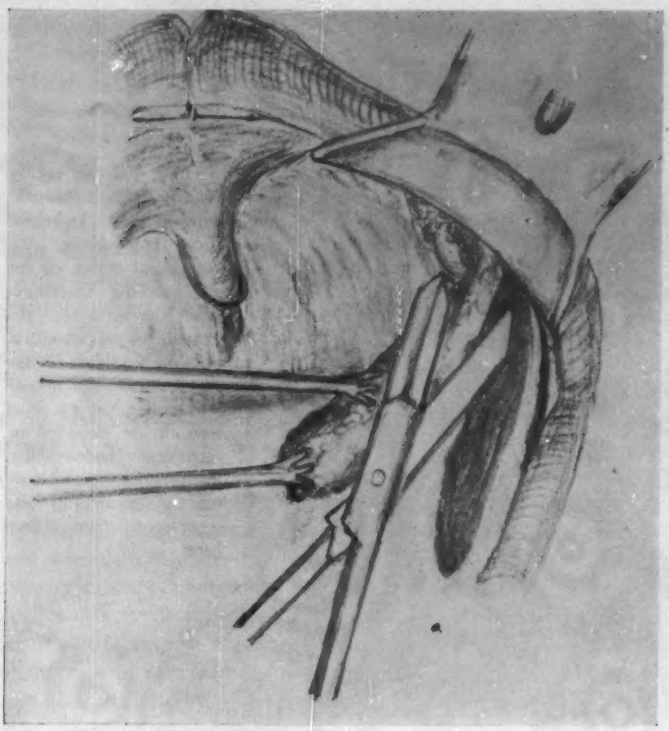

Fig. 5.—Severing upper pedicle of lateral curtain from torus tubarius with ordinary tonsil scissors.

oropharynx to vault revealed by retracting the palate also contrasts sharply with the familiar concepts of the nasopharynx as a small, enclosed space. The total impression is somewhat like an aerial view of a familiar terrestrial landscape. In particular, the tubal prominences appear as small ridges—in marked contrast to their breast-like prominence in the mirrow view. The total impression is initially quite strange and barren. The tubal orifices appear as insignificant dimples. The fossae of Rosenmüller are seen to be rather shallow trenches behind the prominences (Fig. 6). As pointed out by Yankauer,[11] the dimension commonly considered to be the "depth" of the fossa, viz., the distance from the anterior lip of the torus to the lateralmost recess, is rather its length.

After ablation of all macroscopic lymphoid remnants, I have employed the tubal dilators of von Gyergyai, as advised by L. K. Guggenheim,‖ in children suffering either from recurring bouts of otitis media or from conduction deafness. This procedure requires accurate visual identification of the tubal orifices and cannot be carried out blindly.

Adenoidectomy Revision

By far the commonest problem encountered in this procedure is persistence of adenoid tissue due to former incomplete excision. In most of these cases the lateral lymphoid curtains will be found untouched and their resection at the secondary operation presents no special problem. The previous attempt at ablation of the central adenoid mass, however, may result in extensive scarring. Meltzer[7] has graphically described the difficulty which this presents:

The removal of recurrent adenoid growth makes greater demands on the surgeon's skill than a first

‖ References 4 and 5.

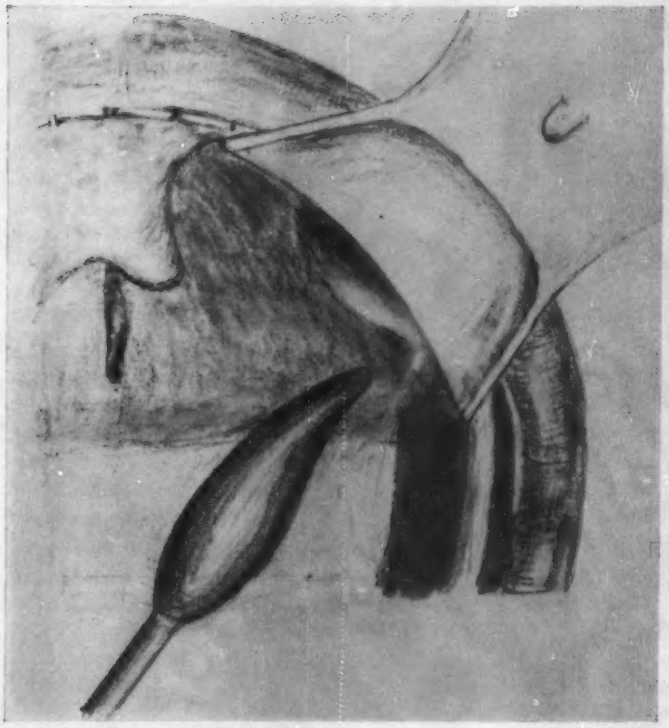

Fig. 6.—Surgically clean lateral nasopharynx, showing left Gyergyai olive approaching the pharyngeal orifice of the corresponding Eustachian tube. The torus is highlighted in an unnatural way for emphasis. Actually it would not appear thus unless the cartilage were to be denuded of submucosa (undesirable). In practice, the structure is considerably less conspicuous than shown here.

operation. Owing to scarring, the tissues may be so firmly adherent to the pharyngeal wall that neither adenotome nor curet can remove them. Even after four or five sweeps into the vault, the adenotome basket may remain empty. This mass of tissue can be removed only by sharp punches. The curet is likely to strip the vault of its membrane and should not be used. Because of the bleeding, second removal is a time-consuming procedure that calls for great care and patience.

I prefer sharp dissection with Allis forceps and scapel or small, right-angled scissors to use of punch forceps in those cases in which scarring so flattens or scleroses the remnants that their engagement with an adenotome is impossible.

A second and less frequent problem is that of true recurrence of adenoid tissue as opposed to "persistence." Meltzer[7] states: "Adenoid regrowth cannot always be prevented. I can state, however, that massive regrowth so common following blind adenoidectomy is very rare following direct adenoidectomy."

It has been found that cases of true recurrence do not, as a rule, present the picture of scarring described by Meltzer.[7] In any event, cases of recurrence are quite amenable to reoperation, even for small collections of lymphoid tissue which are so strategically placed as to interfere with aeration of the middle ears, as over the torior in the Rosenmüller fossae. The mere smallness of the amount of tissue involved is no factor in determining per se a choice between reoperation and radiation treatment.

My experience[1] agrees with Meltzer's ¶ that true recurrences are observed for the most part in allergic children, many of whom have a superimposed suppurative sinusitis. One learns to study carefully the nose and sinuses of all candidates for tonsillectomy and adenoidectomy and to attempt, at least, to control allergy when it is found. Unfortunately, this is not always possible. The allergy may be so aggravated by the

¶ References 6 and 9.

superimposed chronic upper respiratory infection that all attempts at medical management fail to alleviate the condition. Nevertheless, institution of appropriate measures, even before surgery, is not wasted. Skin testing and desensitization are not only quite futile preoperatively but too time-consuming. A nasal smear is made. Even if polymorphonuclear leucocytes appear exclusively, allergy is not considered ruled out and much attention is given to both past and family histories in regard to it. It has been found that so many allergic children are sensitive to foods that I prescribe an elimination diet excluding the commonest offenders: milk, eggs, wheat, fresh fruit, chocolate, nuts, and fish. Environmental control of inhalants is begun at once. Appropriate antibiotic coverage for any infectious sinusitis is employed and has obvious advantages, since wounds are to be created which will inevitably be bathed in pus. At the time of surgery antrum irrigation is not infrequently performed before proceeding with tonsillectomy.

Once removal of tonsils and adenoids has broken the vicious circle of mutual aggravation of allergy and infection, it is possible to study the allergy more at leisure and to use more specific and definitive methods: systematic experimentation with the elimination diet, rigorous environmental control, and, where necessary, skin testing and desensitization.

Meltzer [9] has aptly pointed out that many obscure and ill-understood factors affect the behavior of lymphoid tissue. Allergy is only one. Endocrine influences certainly play a role. A neglected factor of great importance in these cases is the possibility of unresorbed tubotympanal mesenchyme. This embryonic tissue is, unfortunately, not always resorbed at term but may persist for varying periods throughout childhood. The onset of puberty appears to stimulate differentiation into adult fibrous tissue, as does purulent infection. The few failures of direct adenoidectomy may well be due to persistence of this embryonic tissue. L. K. Guggenheim [5] pointed out that exuberant lymphoid growth and persistent mesenchyme may possibly be the result of a single peculiarity of the mesodermal germ layer, and spoke of their interrelation as the "lymphomesenchymal complex." Certainly further investigation of the tubotympanal mesenchyme is needed.

It is worth mentioning that the effect of ionizing radiation (x-ray, beta ray) upon mesenchyme is unknown. An investigation is now underway in our laboratory # to determine whether the effect, if any, is in the direction of promoting resorption or differentiation into adult fibrous tissue—either theoretically conceivable. This uncertainty, coupled with fears as to the safety of the radium nasopharyngeal applicator, has made me eschew this instrument. X-ray has been used for the rare otologic case in which direct adenoidectomy has failed to give a satisfactory result. In some instances this too has failed, possibly because of a deleterious effect—or lack of any effect—on persistent mesenchyme. The only procedure, the effect of which upon mesenchyme is supported by clinical evidence, is dilatation of the Eustachian orifices with the Gyergyai olives, as described by L. K. Guggenheim.*

The worthwhileness of the methods discussed herein can only be evaluated in terms of results. The present paper, devoted to technical matters alone, has avoided any detailed consideration of results. A later publication will consider the problem in those terms.

406 Central Building.

A study of this subject has been underway since 1951 at the Creighton University School of Medicine under a grant from the National Institutes of Health.

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Mobilization of the Stapes Without Incision

A Preliminary Report on Mobilization of the Stapes by Vibrations Originating at the Short Process of the Malleus

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During the past three months, I have been exposing the middle-ear cavity and the incudostapedial articulation in much the same manner as have other surgeons.[1] Once the articulation was visualized, a forked instrument was fitted firmly over it. A rotating, distally flattened rod was brought into contact with the shaft of the forked instrument immediately beyond the ear speculum. This generated vibrations which were transmitted through the crura and the footplate of the stapes to the stapediovestibular juncture and beyond. This technique proved successful in 12 of 18 cases (Figs. 1 and 2). The results thus far have been as successful and gratifying as those obtained with the techniques employed by others.*

The pathway of the vibrations which cause motion of the stapedial footplate originates in the ear canal at the tympanic membrane. These vibrations are transmitted from the tympanic membrane through the ossicular chain to the oval window. Such being the case, it occurred to me that properly directed vibrations of sufficient power and amplitude might be used to break up the otosclerotic focus at the footplate and mobilize the stapes.

It was to be expected that one who was attempting to mobilize the fixed stapes footplate by vibrations directed from the incudostapedial joint would think of other places from which to originate similar vibrations. Since the technique described in the previous article was sound and effective, it occurred to me that similarly induced vibrations might be successfully directed at the ankylosed stapedial footplate from the short process of the malleus. The ossicular chain, in addition to the air within the tympanic cavity, acts as a reducing mechanism. For this reason, it was decided that a much greater vibrating stimulus would be necessary to accomplish the objective—freeing the fixed footplate. Such proved to be the case. During the past two weeks, three patients have been operated on with this technic. In each of the three, there was some degree of improvement. In the first case an average level of 45 db. by pure-tone audiometer was brought up to a level of 15 db. In the other two cases the bone-conduction level would not permit such a striking result; however, there was definite improvement in each.

The idea of transmitting impulses through the ossicular chain with the use of the short process of the malleus as a starting point is not new. In 1884, Lucae[4] described a percussion technic for otosclerosis. He employed a steel rod at the distal end of which was a conical depression. This he fitted over the short process of the malleus. The rod had a spring attached which enabled the operator to deliver short, sharp blows to the ossicular chain. Although Lucae had some success with this procedure, it was abandoned because of the severe pain it induced and because, according to Gruber,[5] it was not successful. In addition, excoriations

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* References 2 and 3. Lindsay, J. R.; Goodhill, V.; Kos, C. M.; Shambaugh, G. E.; Meltzer, P. E.; Fowler, E. P., and House, H. P., in symposium on Operation for Mobilization of Stapes in Otosclerotic Deafness, at the meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Canada, May 17, 1956.

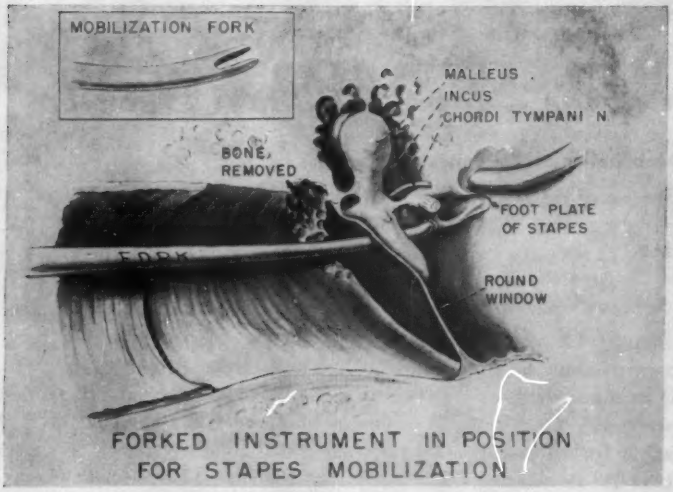

Fig. 1.—Fork placed at incudostapedial articulation.

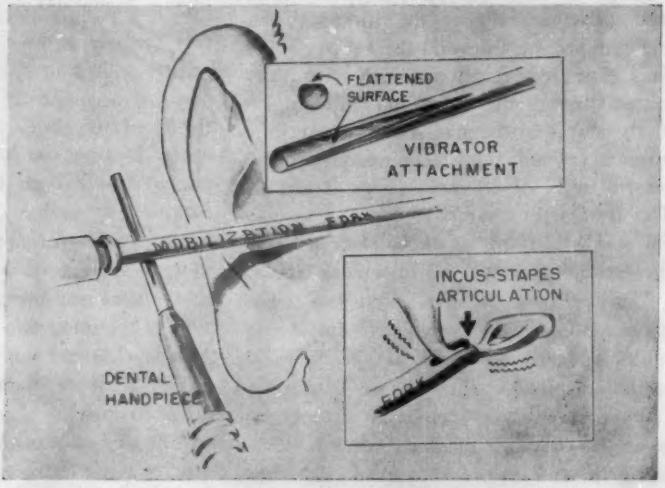

Fig. 2.—Revolving rod in dental handpiece applied to shaft of forked instrument.

occurred on the short process and adjacent tissues and caused pain and discomfort.

The technic of the vibratory attack upon the stapes footplate is as follows. The ear canal is irrigated with warm saline, then with ½ oz. (14 cc.) of aqueous benzalkonium (Zephiran) chloride, 1:1000, just before operation. A 1% procaine solution with 12 minims (0.75 cc.) of epinephrine hydrochloride, 1:1000, to the ounce, is used as a local anesthetic. After preparation of the skin, 0.5 cc. of the solution is injected with a hypodermic needle into the postauricular sulcus at a point corresponding to the posterosuperior quadrant of the external bony canal. Then approximately 1 cc. is introduced using a 25-gauge needle, 1½ in. long. The point of the needle enters the subcutaneous space at the chondro-osseous juncture of the canal. Exposure of the tympanic membrane is obtained with an ear speculum using suitable illumination. Illumination can be by means of a headlight, a head mirror, or an electric otoscope.

The short process of the malleus is identified. A forked instrument especially de-

signed for the purpose is then placed over the short process. The fork is so placed that it is at right angles to a line drawn through three points within the middle-ear cavity. They are the tip of the short process of the incus, the tip of the handle of the malleus, and the lenticular process at the end of the long process of the incus (Fig. 3). This gives the maximum force applied along the axis of greatest vibration in the ossicular chain. When the fork is properly placed, an assistant brings into contact with its shaft a flattened revolving rod in a dental handpiece. The rod rotates

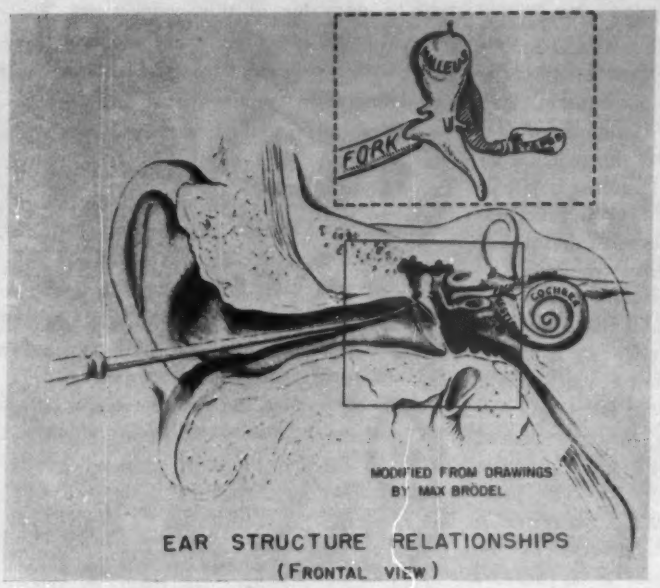

Fig. 3.—Forked instrument placed firmly upon the short process of malleus at right angle to line representing axis of ossicular motion.

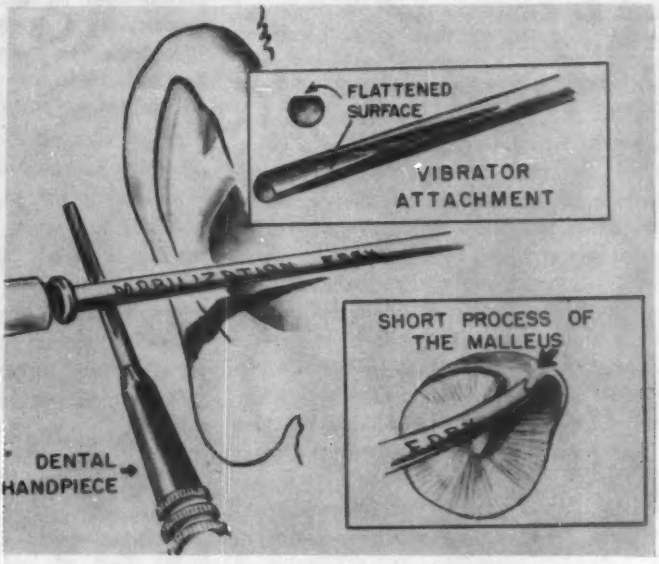

Fig. 4.—Special revolving rod in dental handpiece applied to shaft of forked instrument, positioned upon short process of malleus.

at 9000 rpm (Fig. 4). I have not yet determined the optimal degree of flattening of the distal end of the rod. This will be reported later. Since the impact of the vibrations upon the eardrum is severe, there is some trauma to this structure. As a result of reaction to this trauma, there are extravasation of blood into the spaces of the membrane and slight swelling.† It was noted that these changes took place almost immediately in a patient 78 years of age. In younger patients, 10 to 30 minutes may elapse before redness or swelling occurs. It might be mentioned that the reaction of the tissues of the membrane is far less than that which one encounters when reflecting the tympanic membrane forward or upward for exposure of the middle-ear cavity.

The patients describe the sensation as that of a drill inside the ear. All felt dizzy, but the vertigo was momentary.

I know of no complication which might occur when this procedure is employed.

Experiments on fresh cadavers proved that the malleus could withstand a pressure equivalent to 75 gm. Only sufficient pressure to maintain the forked instrument in position upon the short process of the malleus should be employed. Such pressure is negligible. Too much pressure will dislocate the malleus and defeat the purpose of the procedure.

†If the fork is covered lightly with cotton the trauma is inconsequential.

In a future paper it is my intention to give further details concerning this technic. It is my feeling that this method of attack upon the fixed stapedial footplate will prove to be as effective as other technics now being used. A review of the literature and a report of some of the cases operated on will be of value in this connection. The present paper is a preliminary report which deals with a procedure that has proved safe and effective in the first three cases. I am enthusiastically satisfied that this procedure works; greater experience will enable me to know how effectively.

416 N. Bedford Dr.

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Pharyngomaxillary-Space Abscess

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Deep neck abscesses have presented a serious problem to surgeons for a good many years. References for this paper begin with Dean [1] in 1910, but I am sure the problem had been discussed long before that. It is interesting to compare the literature before and after the use of sulfonamides and antibiotics. Needless to say, the problem is not as difficult as it was before these drugs were available. However, at the County Hospital we know that these abscesses still occur, and I am reporting one here that was seen in private practice.

Discussion of pharyngomaxillary-space abscesses should begin with a short review of the anatomy of the fascia of the neck. The superficial fascia of the neck is that covering the platysma and subcutaneous tissue. Between this layer and the deep fascia is found the external jugular vein. The deep fascia of the neck is divided into superficial and deep layers. The superficial layer of the deep fascia was described by Hall [2] in 1934 as the great cervical sheath which extends from the base of the skull above to the upper end of the thorax below. It is attached to all the bony prominences of the neck, which include the occipital protuberance, the mastoid portion of the temporal bone, the zygoma, the mandible, the seventh cervical spine, the hyoid bone, the scapula, and the clavicle. This layer, referred to by Mosher [3] as the splitting layer, envelops each neck structure, including the sternocleidomastoid muscle, the trapezius, the pretracheal muscles, and the parotid gland.

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The deep layer of the deep fascia of the neck is divided by Hall [2] into the visceral fascia and the prevertebral fascia. The visceral sheath surrounds the trachea, esophagus, and the thyroid gland, and, what is important to this discussion, it forms the external fascial covering for the pharyngeal constrictors and the buccinator. The prevertebral fascia extends from an area between the trapezius and the sternocleidomastoid muscle toward the midline anterior to the vertebrae and meets the corresponding extension from the other side. It extends from the base of the skull superiorly, downward into the thorax behind the esophagus, and splits to form the sheath for the axillary vessels. It forms the posterior wall of the carotid sheath.

One other small but important extension is the alar fascia, which extends from the visceral fascia to the carotid sheath and separates the pharyngomaxillary space from the retropharyngeal space.

The pharyngomaxillary space, also referred to as the parapharyngeal space, is composed of an anterior and a posterior compartment. The anterior compartment is bounded by the buccopharyngeal fascia covering the superior pharyngeal constrictor muscle, the fascia of the internal pterygoid muscle, the medial parotid fascia, the fascia of the styloid and its muscles, the anterior wall of the carotid sheath, and the alar fascia. The posterior compartment is the carotid sheath.

Parapharyngeal infections usually follow infections of the tonsils and pharynx and have followed tonsil surgery, particularly when done with local anesthesia. Brunner [4] reported a number of cases in 1940, among them a case which began as an infection at the base of the tongue, and another which broke through the external canal wall following a radical mastoidectomy. Mosher [3]

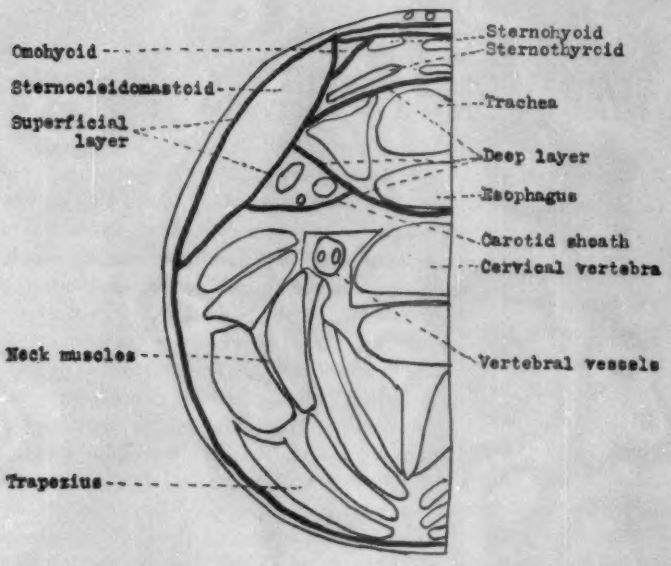

Fig. 1.—Diagram of the layers of the deep facia of the neck.

also refers to infections of the mastoid tip breaking through into the parapharyngeal space, a so-called internal Betzold's abscess.

The pathology, while it may be that of abscess, may be an extension of the infection through the veins with a phlebitis and thrombus formation in the facial and internal jugular veins. Hall [5] reported a number of these cases in 1939 and pointed out the extreme seriousness of these extensions.

The usual picture of pharyngomaxillary-space abscess is a patient who has had a sore throat which may have cleared up several days before pain and swelling developed on one side of the neck. The patient is usually quite ill with a high fever. There is a hard, tender, brawny swelling just below and posterior to the angle of the mandible, sometimes involving the parotid area. There is no fluctuation, and Hall [5] has warned repeatedly against waiting for fluctuation before incising these abscesses. There is a moderate amount of trismus, though this is not as prominent as that found in the masticator-space abscesses. There may be swelling of the lateral pharyngeal wall behind and anterior to the tonsil. If the infection has spread to the posterior compartment, which is the carotid sheath, the neck swelling and tenderness may extend downward along the path of the great vessels and the infection may extend into the mediastinum. If there is not an abscess formation, but thrombophlebitis of the veins, the local signs may be very few, so few in fact as to make diagnosis extremely difficult. Fortunately, antibiotics and sulfonamides have made this problem of diagnosis infrequent now, but prior to the use of these drugs the diagnosis in septic patients was sometimes made at postmortem by finding a thrombosed internal jugular or facial vein.

These abscesses are mainly to be differentiated from masticator-space abscesses reported by Hall and Morris [6] in 1941. The signs and symptoms may be so nearly alike as to make diagnosis possible only when the patient is operated on. Masticator-space abscess is found inside the fascial sling surrounding the mandible and covering the muscles of mastication. It is almost always secondary to dental pathology, and trismus is a very prominent sign.

The treatment of these infections is the administration of antibiotics at once, the application of hot compresses externally, and incision as soon as there is reason to believe an abscess is formed. The abscess

is deep, and fluctuation will not occur. Prior to the use of antibiotics, early ligation and excision of the facial and internal jugular veins was often lifesaving and still is indicated if the patient is not doing well and there is a blood-stream infection.

The incision of these deep neck abscesses may not be easy with landmarks obliterated by marked swelling. Mosher[3] in 1929 reported his submaxillary approach, in which he makes a T-incision over the submaxillary gland, elevates the gland, and then, using the great horn of hyoid bone and the styloid process as landmarks, carries the incision down or up with blunt dissection. We have usually made an incision parallel to the anterior border of the sternocleidomastoid muscle and, using the angle of the mandible, the anterior border of the sternocleidomastoid muscle, and the styloid process as landmarks, have carried the incision downward with blunt dissection to the abscess.

Brunner[4] has incised some of his cases through the pharyngeal wall from the inside. This depends on where the abscess points, but our experience has been that an external approach is almost always indicated.

The following case is reported as an example of a pharyngomaxillary-space abscess developing over a period of time, during which the patient had been receiving antibiotics.

Report of a Case

A 52-year-old male office worker was referred with a diagnosis of left mastoiditis and an x-ray report of cloudiness over the left mastoid antrum. He had had swelling in the left neck for two weeks which had been getting more painful. He had received penicillin and streptomycin sulfate injections daily during this time and had had an occasional temperature of 100 F. There was no history of sore throat or upper respiratory infection.

Twenty-five years ago he developed osteomyelitis of the right hip and has had a draining sinus from that region since that time. Six years ago he had an osteomyelitis of the left mandible which drained for several months. Five years ago he had an operation on his left tibia for chronic osteomyelitis, after which this wound healed.

Examination showed a rather stout man who walked with a limp. Temperature was normal. Both tympanic membranes were normal. There was a hard, brawny, tender swelling below and posterior to the angle of the left mandible, including the lower parotid region. There were old scars over the ramus of the left mandible. There was only moderate trismus and no swelling of the left lateral pharyngeal wall. There was a draining

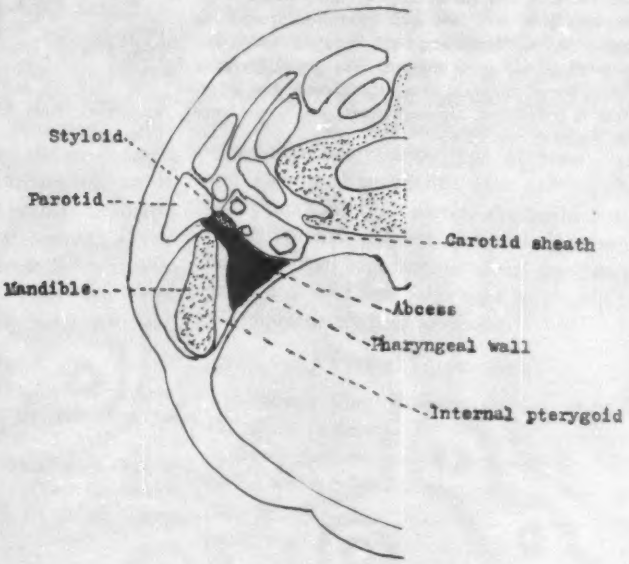

Fig. 2.—Diagram showing cross section through the abscess.

sinus in the right hip region, the right leg was shorter than the left, and there were old scars over the left tibia.

The patient was started on chlortetracycline (Aureomycin), 500 mg. q. i. d., and the following day was improved, with slightly less swelling and less pain. Temperature was normal. On the second day pain was increased again, temperature was 100 F, and there was a slight swelling in the left pharyngeal wall. The patient was hospitalized, and with heavy premedication and local anesthesia (2% procaine hydrochloride) an incision was made high along the anterior border of the sternocleidomastoid muscle, through the superficial layer of the deep fascia. Then, using the angle of the mandible, the anterior border of the sternocleidomastoid muscle, and the styloid process for landmarks, the incision was carried with blunt dissection to the carotid sheath, then turned inward under the mandible toward the tonsil and the pharyngeal wall, where a large abscess was found. A Penrose drain was inserted. Postoperatively the patient was slow to recover, but with hot compresses, chlortetracycline, and penicillin the drain was removed in one week, and three weeks after incision the wound was healed.

X-ray of the cervical spine three weeks after the beginning of symptoms was negative. Culture from the abscess showed hemolytic Staphylococcus. Culture from the hip sinus showed only contaminants and no hemolytic Staphylococcus.

The source of this abscess is not clear. There was no history of any pharyngeal infection for months before pain and swelling began in the neck. There were seven lower front teeth, four on the left and three on the right, which were in very poor condition with ulceration around the roots. All the remaining teeth were out, and x-ray showed no old roots present. The possibility of a blood-borne infection from the chronic hip infection is considered, although there was no evidence of sepsis at any time.

Summary

The anatomy of the fascia of the neck is reviewed with its relation to the pharyngomaxillary space. The etiology, pathology, signs and symptoms, and treatment of pharyngomaxillary-space abscess are discussed. Under treatment it is particularly emphasized that these abscesses are deep and that we should not wait for fluctuation before incising them. A case is reported of a 52-year-old man who had had repeated recurrences of chronic osteomyelitis and who had been on penicillin and streptomycin sulfate for two weeks before incision and drainage of the abscess. A hemolytic Staphylococcus was cultured from the abscess.

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Hypoadrenocorticism in Otolaryngology

Treatment with Whole Adrenal Cortex Extract

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Many problem patients in otolaryngology are treated in diverse and often unsatisfactory fashions because of vague symptoms which are considered to be emotional in origin and are frequently disregarded. With an understanding of certain endocrinologic precepts, the basis of many of these complaints can be put on a definite physiopathologic etiology. These patients react poorly to stress situations because of a lack of adequate hormonal production or an imbalance among these hormones.

In a previous communication [1] the various types of hypoadrenocorticism were classified as to their origin. Regardless of how the hypoadrenocorticism developed, we have a typical type of patient presenting many common symptoms. These are the patients who are usually very meticulous in their work: perfectionists who drive themselves to exhaustion, since they know not the meaning of the word relaxation. They often seek the assistance of the psychiatrist and more than likely are thrown into an ill-defined group of schizophrenia or manic depression. A common statement is that they had been previously treated for anemia and low blood pressure. They are often misguided because of a low basal metabolic rate which had been passed off by the physician as the classic example of the erroneously described atypical hypothyroidism found in most textbooks. They have further had the unfortunate experience of an aggravation of their symptoms when placed on intolerably high dosages of thyroid extract.

From careful observation most of their complaints were found to be similar to those recognized in hypoglycemia. These patients are nervous, irritable, and apprehensive. They frequently complain of excessive weakness, fatigue, depression, faintness, and insomnia, these being most pronounced following physical, emotional, or infectious stress. Epigastric distress is frequently present, and many of these patients have had repeated x-ray studies in an attempt to demonstrate a gastric ulcer. Alternating bouts of diarrhea and constipation have led to investigations of the lower bowel in a futile attempt to discover the pathogenesis is ulcerative colitis [2]: The menstrual history has its onset later than average. Premenstrual tension is marked, manifest by bloating of the abdomen and breasts, nasal congestion, migrainous type of headaches, and emotional instability with craving for candy and salt. However, the periods are regular though profuse and prolonged, with midmenstrual slowing on about the fourth or fifth day. These facts may be elicited by a competent medical assistant.

Physical Appearance

Although these findings seem to wander far afield from our specialty, we must keep in mind that we are dealing with the problems which have not responded to our ordinary therapeutic repertoire. Therefore, we must try to uncover the basic functional

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etiology of these ear, nose, and throat symptoms which bring the patient to the otolaryngologist. With a little experience it is possible to recognize even on casual observation that these patients are deficient in their output of adrenal cortical hormones.

These patients are of the thin, asthenic body habitus, have sparse body hair, thin dry skin, and a long thin neck, bruise easily due to a capillary fragility, appear apprehensive, are observed to have a cold, clammy perspiration of the face, with color draining easily from their lips, when subjected to minor stress, and have a tendency to faint easily.

A confusing factor with sinusitis may be the marked puffiness of the lower lids upon arising in the morning. In children this finding requires a check of the urine for albumin because of the possibility of nephrotic involvement. There are many allergic manifestations found, including urticaria, angioneurotic edema, hay fever, asthma, eczema, atopic dermatitis, and other skin lesions.

They usually have an abundant head of fine hair; often a dilatation of the pupils (with an autonomic instability); typical denture formation, with crowding of the lower incisors; generalized cervical lymphadenopathy; eunuchoid manifestations of the hands, in that the index finger is longer than the fourth finger; women are frequently flat-chested; men have sparse chest hair; the extremities are proportionately longer than the torso, and there is an absence of hair on the lower lateral two-thirds of the legs.

Physical Findings

In a great percentage of cases of hypoadrenocorticism an allergic conjunctivitis was found, accounting for blurring of vision, squinting and tearing, and crusting of lids. Nasal findings include injection of the septal mucosa and a pale-pink or light-violet color to the mucosal membrane of the enlarged turbinates. These patients therefore complain of nasal stuffiness. They also complain of a scant, thick, mucoid discharge, which they find difficult to dislodge from the pharynx. There frequently is an injection of the pharyngeal mucosa with some thickening of the lateral bands unconnected with any sinus pathology. In the physical examination there is found a generalized cervical lymphadenopathy, including submaxillary, posterior cervical, and jugular chains. The referring physician may have informed us that the patient has a droplet-shaped heart, possibly some fine crepitant or sonorous rales at the base of each lung (which is on an allergic basis), a generalized visceroptosis, an orthostatic hypotension, and a positive Rogoff's sign (tenderness when pressure is applied to the adrenal area at the junction of the lower rib and the erector spinae muscles).

Examination of the blood reveals a relative lymphocytosis, hemodilution but with a color index of 1, and an eosinophilia. Urinalysis is usually negative but may show a slight albuminuria.

Of all diagnostic tests for hypoadrenocorticism, we consider the four-hour glucose tolerance the most important. Of significance is the low fasting blood sugar, with a slight rise after one to two hours and a subsequent fall to hypoglycemic levels. In older persons with a history of dietary indiscretion (particularly of alcohol) and other intoxications, including quinacrine (Atabrine) and heavy metals, a hyperglycemic level may be reached during the first half-hour or one- to two-hour determination. Complicated and expensive laboratory tests are unnecessary.

Otolaryngologic Applications

The knowledge of hormonal dysfunction can be applied to the treatment of a considerable number of otolaryngologic conditions, many of which have resisted standard forms of therapy. Proetz[3] has indicated the importance of thyroid insufficiency in nasal pathology in a series of cases reported in 1948 and 1950. Application of these endocrinologic principles opens a new vista

in the relief of these heretofore regarded as unimportant but extremely distressing and aggravating conditions, so that patients may enjoy the full bounty of normal health. These are the patients who would return continuously with the same symptoms despite the nasal tampons and various throat gargles and sprays and antibiotics.

Allergies

Assuming that allergic conditions are considered to be on the basis of an adrenal cortical insufficiency, a new method of approach can be added to the treatment of one of the commonest disorders encountered in practice. We are treating the patient as a person and not treating him symptomatically, so that the over-all result should be most gratifying. Apart from seasonal and other respiratory allergies, we have noted in this particular type of person that when he is subjected to stress there is a paling of the nasal mucosa, a blockage of the nasal passages, an increased mucoid secretion, and difficulty in nasal breathing. Emotional stimuli or infections can deplete the adrenocortical reserve, resulting in bronchial asthma. Hansel,[4] in his treatment of allergy with the use of infinitesimal dosages of antigen, theoretically and in actuality, should obtain the desired results because the nonspecific protein reaction does not stimulate the adrenal beyond its reserve, and at the same time builds up antibodies to neutralize the specific allergen. The ordinary high dosages may stimulate the pituitary-adrenocorticotropic hormones to such proportions as to exhaust the adrenal reserve. Concomitant treatment with whole adrenal cortical extract * affords a greater reserve of adrenocortical action, in contradistinction to steroid therapy, as is the vogue today in the use of cortisone, hydrocortisone, prednisone (Meticorten), etc., which in actuality causes greater adrenocortical suppression and even atrophy, rendering the patient more incapable of withstanding unexpected stress. Adrenal cortical extract in the amount given is not a replacement therapy but provides a physiologic proportion of adrenal steroids to temporarily suppress adrenocorticotropic activity of the pituitary, allowing the adrenal to rest and thereby permitting normal function of the gland to be regained. In other words, treatment with whole adrenal cortical extract aims at restoring normal function, while individual steroids give dramatic but temporary relief and at the same time may be causing irreparable damage.

Vasomotor Rhinitis

Many patients with no typical positive tests to allergies have been diagnosed as having vasomotor rhinitis, but these have proved to be typical cases of hypoadrenocorticism and respond quickly to adrenocortical therapy and an autonomic nervous system stabilizer.† The diagnosis and treatment of most endocrinopathies is facilitated by having a sound understanding of the two great integrating systems of the body—the nervous and the hormonal systems—and the part they play in our concept of stress.

At times by the simple expedient of limiting carbohydrates in children we have eliminated the distressing symptoms of nasal stuffiness. Readily absorbable carbohydrates (including alcohol)‡ in any form act as a definite and violent stresser agent on the adrenal cortex.

Gonadal dysfunction is also known to cause a vasomotor instability, so that adjunctive estrogens or androgens may be indicated, which also relieve the adrenocortical production of these particular hormones, allowing the adrenals to regain their reserve. This hypothesis explains the inability of otolaryngologists to relieve nasal congestion of women during the third trimester of pregnancy, at which time the hypophysial action neutralizes estrogenic production.

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* Eschatin (Parke, Davis & Company, Detroit).

† Bellergal (composed of belladonna alkaloids, ergotamine tartrate, and phenobarbital) (Sandoz Pharmaceuticals, Hanover, N. J.).

‡ References 5-7.

Headaches

The patients who come to the otolaryngologist have been thoroughly investigated from a medical standpoint by their family physician and found wanting as to the diagnosis, and therefore their disturbances are considered to be functional, cranial, or otitic in origin. Many present themselves after having withstood the ravages of shock therapy, encephalograms, and psychoanalysis and having run the gamut of all the barbiturates and narcotics.

Although not in the province of otolaryngology, they are directed to us because of these dull, throbbing, persistent frontal or occipital headaches with no visible ear, nose, or throat pathology. Here, again, the otolaryngologist is expected to be a medical diagnostician and not only an otolaryngologic technician. These are not typical tension-type vascular or histamine headaches but often are caused by spasm in the sternomastoid and trapezius muscles, which is one of the common symptoms found in hypoadrenocorticism. Nor are these to be confused with the sphenoid sinus type of headache or Thornwald's bursa in the nasopharynx. Palpation of these muscles elicits excruciating tenderness and spasticity, only temporarily responsive to procaine injections or oral mephenesin. Nasal stuffiness may be a concomitant finding.

Premenstrual tension headaches may be due to an imbalance in the electrolyte control by the adrenocortical steroids producing a retention of intracellular potassium, with a resultant increase of fluid within the cells. Many patients with typical migraine headaches may have gonadal and adrenal dysfunction, which should be investigated.

Virus Infection

We have all noted the persistent nosebleeds, lassitude, sleepiness, and extreme tiredness following viral infections, and our observations have been that the most aggravated symptoms are found in the hypoadrenocortical cases. The latter condition presents increased capillary bleeding, especially in the influenzal type of infection, and the weakness can be accounted for by the depletion of any cortical reserve the patient may have had. In severe viral infections even patients with an intact adrenal may complain of such symptoms, which may be alleviated by minimal doses of adrenal cortical extract. This is of particular significance in shortening the period of convalescence in industrial medicine. Also, with cortical depletion we expect hypoglycemic episodes, at which time the patient is accident-prone, involving errors of judgment, mental efficiency, and manual dexterity.

An annoying situation is the patient who is seen frequently in the office with recurrent sore throats and "colds." There is no evidence of infection of the lymphoid tissue of the pharynx or nasopharynx, the sinuses are negative, and there is no vocal pathology. These patients have no resistance to repeated viral infections, do not respond to antibiotics, and, in contrast, respond well to hormonal and antihistaminic therapy.

Ménière's Disease

Many cases of Ménière's disease have been designated as functional and due to psychogenic stress.§ The authors in two distinctly different fields of medicine have had many cases referred to them with this diagnosis. The glucose-tolerance tests have frequently shown drops in blood sugar to hypoglycemic levels, which, when corrected, are very frequently followed by recovery from the dizzy spells.[11] More extensive investigation was instituted to determine the integrity of the vestibular system and middle ear. When no obvious pathology had been found in the middle ear, nasopharynx, or nose and no otologic etiology could possibly be elicited and treatment of the basic hypoglycemia was instituted, results were uniformly excellent. Therefore, the glucose-tolerance test is required of any patient complaining of dizziness and suspected of having Ménière's disease.

Hilger[12] believes that the symptoms are

§ References 8-10.

due to autonomic dysfunction in the labyrinthine neurovascular sphere. "The vascular anatomy of the labyrinth is ill-designed for the resorption of the transudate resulting from ischemic damage to the capillary due to angiospasm." He believes that there is a gonadal deficiency and an additional stress factor pulls the trigger to initiate symptoms. This is in accord with our explanation that stress depletes adrenal cortical reserves, causing symptoms when adaptation has reached the stage of exhaustion.[13]

Menkin [14] demonstrated that adrenal hormones decrease capillary permeability and normalize it. In this respect a combination of ascorbic acid plus bioflavonoids ‖ shows a similar activity and has been used successfully, primarily in protecting the capillary system against damage.[15] A normal capillary system is well adapted to meet the emergency calls imposed by the stress reactions. But once the capillary wall is altered and increased capillary fragility is evidenced, the capillary system can no longer stand the burden of the stress reaction. Adrenocortical therapy leads to repair of the capillary system and the resorption of the transudate and diminishes the tendency to angiospasm. Gonadal therapy relieves the vasomotor instability through the autonomic nervous system and is further enhanced by an adjunctive autonomic nervous system stabilizer.

Surgery

From our studies of these interesting phenomena, as described in a large series of patients,[1] and recently described in an article on otolaryngologic surgical procedures,[16] we have evolved some observations of seeming importance to the surgical patient. We studied the effects of administering supplemental adrenal cortical extracts to those receiving general anesthesia (children) and some patients receiving local anesthesia (adults). Amelioration of surgical as well as psychologic trauma was our goal.

It was found that a certain percentage of patients, although not seriously ill, were ill-equipped physiologically to withstand the emotional impact of surgery, or even its anticipation. We described the preparation of these constitutionally inferior persons so that they could withstand the stresses and rigors of surgical procedures with a minimum of adverse reactions.

Any surgical or stressful procedure calls for an adequate supply of adrenocortical hormones to meet the increased needs for cellular activity. It is generally agreed that sudden demands may often be enormous compared with ordinary daily physiologic needs, and the ability of the adrenal to respond to these demands varies from person to person. There is a host of different hormonal elements, originating in the adrenal cortex, which are essential for resistance to stress. There is a feeling of well-being which goes with increased nourishment of tissues by the glucosteroids. There is an enhanced electrolyte balance, as well as several other types of metabolic modulation from other components.

From much of the literature, it would appear that intravenous hydrocortisone (and some of its newer modifications) is the agent of choice during surgical procedures because "approximately 80% of the cortical steroids found in adrenal venous blood is hydrocortisone."[17] With infusions of hydrocortisone, however, side-effects are common, particularly if surgery is prolonged. If as little as 50 mg. of hydrocortisone is given, adrenocortical suppression is imminent. Changes in the adrenal may persist for six months, or permanently, after prolonged hydrocortisone therapy has been terminated.¶ It is established, however, that the amount of cortisone-like activity in 10 cc. of adrenal cortical extract is of the order of 1 mg. of cortisone and this amount, or 10 times this amount, cannot suppress adrenocortical activity and has no adverse effect, as mentioned above. Adverse psychological reac-

‖ C.V.P. (U. S. Vitamin Corporation, New York).

¶ References 18 and 19.

tions are commonly encountered in the use of hydrocortisone, but we have never had, nor have we been able to learn of, any but tranquilizing effects with the use of whole adrenal cortical extract. We have contended that there is a physiologic balance of hormones in the adrenal cortical extract, whereas the use of a single member produces an imbalance in the glandular complex. It must be assumed that there is a synergistic relationship between the several components enabling each to exert a far greater effect when in this natural physiologic ratio than when given alone. Recently, Selye[20] has succeeded in demonstrating that aldosterone, which has a close chemical relationship to desoxycorticosterone, antagonizes cortisone-like compounds, verifying the theory of balance between two opposing types of corticoids.

Treatment

General Principles

In general the treatment is such as outlined for surgery, but there are certain special conditions requiring minor modifications. In all of these we believe that proper adjunctive measures with reference to the adrenal cortex are paramount. The primary aim here is to include therapy which will relieve the adrenal cortex of excess stress and to give it an occasional "refractory period" in order to allow the cells to return to a better functioning state. Sufficient amounts of adrenal cortical extract are given so that the target organs, depending upon these hormones, are adequately supplied to produce a normal physiologic response.

Although whole adrenal cortical extract is possibly a main implement of our armamentarium, it is very definitely only a part of the therapy. There is never a situation of a pure glandular dysfunction without the involvement of the entire glandular system.[21] For example, many ramifications are seen in an upset of the thyroid adrenal axis. Ultimate improvement of one gland may be accomplished through extracts of another. It is often vitally important to utilize adjunctive hormonal therapy in varying amounts and combinations to correct specific metabolic aberrations. Thus we found use of parathyroid hormone, estrone, testosterone, thyroid, progesterone, etc., often an essential link in a successful completion of our therapy. An autonomic nervous system stabilizer, Bellergal, has been of value in obtaining this integration and stabilization.

We cannot overemphasize the importance of diet in conjunction with therapy. Patients will usually adhere to the prescribed diet once they realize from careful discussion that dietary indiscretions will cause an exacerbation of their symptoms. In general, the diet consists of the strict elimination of rapidly absorbed carbohydrates in order to obviate sudden rises in blood sugar with subsequent falls. Such food stuffs are considered "adrenotoxic." Between-meal feedings of milk or fruit are advised to prevent any slackening-off of blood-sugar levels, which are prone to occur two to three hours after eating. Salt is allowed in unrestricted amounts because of a tendency to sodium depletion. A diet high in protein, moderate in fat, and low in carbohydrates is advised as follows:

FOODS ALLOWED

All meats, fish, and shell fish
Dairy products (eggs, milk, butter, and cheese); also recommended: 1 pint to 1 quart acidophilous milk daily
Milk between meals; milk, cheese, and/or butter and saltines before retiring
All vegetables and fruits not listed below
Salted nuts (excellent between meals)
Peanut butter
Protein bread
Sanka, weak tea, and sugar-free sodas
Soybean and soybean products
Cyclamate calcium (Sucaryl) as a substitute for sugar

FOODS TO AVOID

Potatoes, corn, macaroni, spaghetti, rice
Pie, cake, pastries, sugar, candies
Dates and raisins
Cola and other sweet soft drinks
Coffee and strong tea
All hot and cold cereals (except occasionally oatmeal)

Specific Applications

1. Allergies.—Very dramatic but temporary results are obtained with individual

adrenal steroids, but more complete and lasting results are obtained through adrenal cortical extract and dietary therapy. Of such significance is the strict adherence to the dietary regimen in this condition that we are able on precursory examination of the nasal passages to determine whether the patient has been strictly adhering to the diet or not. Ordinary immunologic procedures may also be carried out but are recommended in small dosages.

In severe allergic cases it may be necessary to hospitalize the patient for treatment. Adrenocortical extract in 10 cc. doses every four to six hours may be required for the first day or two. With the relief of symptoms, the intervals may be increased to 8, 12, and 24 hours, then biweekly, etc., until optimal maintenance or dietary control alone is found adequate. Minimal doses of thyroid extract, estrogens, or androgens may be indicated, especially in patients presenting symptoms of the climacteric.

2. Vasomotor Rhinitis.—Again, strict limitation of carbohydrates and alcohol is of the utmost importance. The general principles of treatment of hypoadrenocorticism are applicable. Thus a great percentage of patients are dramatically relieved, but in the remainder additional medications may be necessary. Bellergal, from one to six hours daily, is often required. Combinations of estrogens and androgens in the ratio of 10:1 are invaluable adjuvants in both men and women.

3. Headaches.—Since the patient may not be seen until late in the course of his condition, he may prove difficult to treat because the psychic viewpoint has been so strongly emphasized and followed up. Shock therapy, if unsuccessful, further depletes an already damaged adrenal cortex. Even with the use of barbiturates or narcotics the weaning process is made much simpler through adrenal cortical therapy and at the same time we are correcting the underlying etiology. Therapy alleviates the tension of the sternomastoid and trapezius muscles, relieving the hypoadrenocortical patients of their headaches.

4. Virus Infections.—Viral infections with their otolaryngologic symptoms are not amenable to most antibiotics. Concentrated adrenocortical extract in oily solution,# having a prolonged effect, is more efficacious in this type of condition. Only one or two weekly injections are necessary for the usual acute attack, but more prolonged therapy is needed for the recurrent infections.

5. Ménière's Syndrome.—Intravenous adrenocortical extract should be given frequently until the patient has been relieved of the acute symptoms. Diphenhydramine (Benadryl) given intravenously helps relieve concomitant nausea and any associated allergic manifestations. Bellergal stabilizes the disrupted autonomic nervous system. The slightest variation from the prescribed diet results in recurrence of symptoms. Anticipated stress requires prophylactic injections to avoid an attack.

6. Surgery.—Prior to any surgery we should consider the necessity of supplemental adrenocortical hormonal therapy, intramuscularly or intravenously. Adrenocortical extract may be required prior to, during, or following surgery, or at all of these times. There is no need for concern over the electrolyte and water balance of the body. If indicated, however, dextrose or saline may be given as desired, with doses of adrenocortical extract ranging from 5 to 200 cc. In children, excellent results have been obtained with as little as 2.5 cc. prior to surgery and 2.5 cc. upon completion of surgery. The child who receives adrenocortical extract appears much more relaxed under anesthesia and requires a smaller amount of ether to keep it at a surgical anesthetic level; also there is an obvious tranquilizing postoperative effect.

Comment

A syndrome of hypoadrenocorticism and its ramifications in several types of path-

Lipo-adrenal cortex (The Upjohn Company, Kalamazoo, Mich.).

ology and stress conditions has been further explored. Its particular application to the problem of otolaryngology is important to all of us, since the adrenal cortex plays a vital role in maintaining homeostasis in many abnormal conditions involving stress. The use of the whole adrenal cortical extract in the treatment of conditions allied with adrenal insufficiency has been neglected since the development of the adrenocorticotropic hormone (corticotropin [ACTH]) and cortisone. It must be pointed out that these latter agents produce dramatic and permanent results only in such acute conditions as, for example, delirium tremens, certain ophthalmologic diseases, and other emergency crises, but the results are only temporary and even dangerous in chronic diseases. Many will argue that the amount of beneficial glucocorticoids in 10 cc. of adrenal cortical extract is minimal and of about the order of 1 mg. of cortisone-like activity. Much work is indicated to establish why such good therapeutic results are experienced with this amount than that predicted on its cortisone-like activity. It has been postulated that there is a synergistic activity between the 30-odd hormonal elements of natural adrenal cortical extract (ACE) and, further, that still other therapeutic and protective elements elaborated by the adrenal cortex are, as yet, unidentified. We know, also, that the adrenal cortical extract elaborated by a gland stimulated by corticotropin is not of the same composition as that found in the natural gland. Corticotropin is almost universally viewed as the tropic hormone for cortisone. One must ask, however, if it is, also, to be considered the tropic influence for the other steroids and which of these appear as a physiologic response to the various needs of the body.

Summary

A description of a typical hypoadrenocortical patient is outlined in order that the otolaryngologist may quickly and easily recognize such a person. Further physical and laboratory tests are suggested to corroborate this diagnosis. Several important but previously slighted ear, nose, and throat conditions are discussed in their relationship to adrenal cortical insufficiency. A more successful otolaryngologic approach to Ménière's syndrome, vasomotor rhinitis, allergies, headaches, virus infections, and surgical procedures is suggested through the use of the whole adrenal cortical extract. Special emphasis is expressed regarding corticotropin (ACTH), and cortisone and its derivatives in their production of damaging and deleterious side-effects. An aqueous extract of the adrenal cortex is a physiologically balanced mixture of all adrenal steroids, which, by allowing for a refractory period, increases the ability of the patient's own adrenal cortex to respond normally. By thus treating the whole patient and not any limited disturbance, more prolonged and lasting results are obtained. With recognition of the basic glandular dysfunction instead of treating local tissue pathology the otolaryngologist attains more satisfactory therapeutic results.

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Labyrinthine and Intracranial Lesions

The Value of Mass Caloric and Turning Tests in Neurotologic Diagnosis

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It is my purpose in this presentation to point out the necessity of doing the mass caloric and turning tests for a more complete diagnosis, since there has been a tendency of late to rely mostly upon minimal caloric tests alone.

In reviewing a fair number of neurological, neurosurgical, and neurotologic cases at the Temple University Hospital and the Albert Einstein Medical Center, I observed the following facts:

The minimal tests or the modifications of the original Kobrak tests are excellent for screening a large number of persons, but in pathological cases the mass caloric tests are the best choice, and for obvious reasons.

In neurotologic diagnosis we depend a great deal upon the vegetative nervous system symptoms, such as pallor, perspiration, nausea, vomiting, etc., and, to a lesser extent, upon past-pointing in the differential diagnosis between a supratentorial and an infratentorial lesion. We also depend upon a longer period of ear stimulation in moderate and severe impairment of vestibular function to elicit the additional normal, diminished, exaggerated, or perverted vestibular phenomena of nystagmus, vertigo, falling, and past-pointing.

The minimal caloric tests do not supply the adequate stimulus in many pathologic conditions.

One can easily convince himself of this fact by using the minimal and mass caloric tests in a small series of cases in which the vestibular function is moderately or severely impaired.

The minimal tests, unfortunately, are in a very chaotic state at present. There is no standard, they have a limited application, and in abnormal cases they are more time-consuming, since the tests have to be repeated several times.

Some otologists use the quantity of water at a given temperature as their standard, which may vary from 1 to 50 cc.; others use the temperature of the water as a standard, ranging from 89° to 86° F to 68° to 65° F to 55° to 42° F and ice-cold water, 32° to 34° F. Still other otologists use the period of time used to irrigate the ear as their standard.

The application of the tests likewise varies. Some otologists insist upon strict pessimum and optimum positions while caloric tests are performed; others instill the water into the ear with the head on the opposite shoulder, followed by turning the head to the same shoulder to allow the water to drain out of the ear being tested, followed by righting the head into erect position, which, in turn, is followed by the pessimum and optimum positions. The instillation is done by some with a medicine dropper; others insist upon using a cannula attached to a Luer-Lok syringe and a stream of water directed under vision through an ear speculum toward the posterior-superior canal wall. Occasionally the cannula may become detached and injure the drumhead; or, if it is held at a distance from the drumhead, part of the water will be lost by not reaching the area to be chilled or warmed, as the case might be.

Again, some otologists observe the latent period from the beginning of the applica-

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tion of the stimulus to the onset of nystagmus; others observe the latent period to the termination of the nystagmic movements of the horizontal canals; still others omit the observation of the latent period entirely.

A minimal stimulation will elicit a minimal response, which can easily be overlooked unless nystagmography is used, which is prohibitive economically to otologists and patients alike. Ice-cold water will blot out minimal responses. The hearing tests prior to the advent of a standard audiometer were in a similar chaotic state; no two otologists used the same pitch and intensity in speech testing, nor did they use the same standard in the watch-tick, coin-click, and Galton-whistle tests. The tuning-fork tests varied in calibration and mode of excitation; the higher frequencies could not be tested. The audiologist today can not plot the various audiometric curves of hearing impairment of air- and bone-conduction loss in otosclerosis, determine the gain following fenestration or stapes mobilization surgery, or determine the suitability of a hearing-aid fitting by means of a 5-10-15 db. intensity stimulus. The audiologist requires the full scale from 5 to 100 db. Why should the neurotologist expect to be able to evaluate the moderate to marked vestibular mechanism dysfunction by means of a minimal stimulus? The mass caloric tests can be likened to audiometry. In the mass caloric tests we employ a large container and large amount of water, but we use only a minimal amount of water in normal persons or in persons with a mild degree of deviation of function from the normal. In cases of moderate to severe vestibular mechanism function loss we have more water to use as maximal stimuli up to five minutes and with no need of repetition of tests and no need for a waiting period for the vestibular reflex to recuperate from fatigue, thereby limiting the caloric tests to five minutes in total loss of labyrinthine function.

The standard Bárány caloric tests accomplish this purpose well: The physiologic pessimum and optimum positions are adhered to, the cold-water (tap) temperature is 68 F, and the end-point of nystagmus is not difficult to measure. When the irrigating volume is about 100 cc. per minute, the entire drumhead and the posterior-superior canal wall are chilled or warmed and there is no need for irrigation by means of a cannula under direct vision.

The other phenomena which are helpful in the differential diagnosis, such as vertigo, past-pointing, and, above all, pallor, perspiration, nausea, and vomiting, are more often elicited upon maximal caloric stimulation. Spontaneous nystagmus is inhibited or reversed upon mass caloric but not upon minimal caloric tests.

Jongkees states that the frequency of the nystagmus varies with the repetition of the minimal tests, a common practice employed if the first minimal tests fail to elicit vestibular phenomena.

Many believe that 5-30 minutes should elapse between minimal tests if repetition of tests is necessary. The latency of responses, according to Cawthorne, Fitzgerald, and Hallpike, varies with the thickness of the bony structure of the petron, hence more stimulation may be necessary.

One questions the wisdom of introducing the minimal tests into neurotology at the expense of diverting valuable time and labor from more important research problems. There is nothing to gain time-saving-wise and certainly quite a bit to lose as far as correct diagnosis is concerned.

The standard mass caloric tests were the tests used by Bárány, Fisher and Jones, Grant and Fisher, McKenzie, and others, who worked out the different syndromes which are suggestive or indicative of the different intracranial and peripheral lesions, most of which were verified by surgery, autopsy, or competent neurological examinations and opinions.

Another very important part of the vestibular examination is the turning tests. I admit that many of the cerebellopontile-angle lesions can be diagnosed by the caloric tests alone in the presence of the spontane-

ous pathological phenomena, such as pathological nystagmus, ataxia, past-pointing, and, above all, evidence of a central nervous system lesion.

However, there are atypical cases and lesions which extend into the fourth ventricle or bilateral lesions which can be diagnosed with more certainty when the turning tests are also done, namely, when the absence or presence of markedly subdued vestibular phenomena is observed.

In frontal lobe lesions simulating cerebellar lesions the turning tests may elicit prolonged past-pointing with either arm to either side, suggesting motor cortex involvement. They may also elicit vegetative nervous system phenomena which may not be brought out by the limited caloric stimulation alone—findings which most often rule out a cerebellar lesion.

Exaggerated and prolonged vestibular responses are brought out chiefly by turning tests.

Exaggerated nystagmus revealed by turning tests is suggestive of the following lesions: pituitary, suprasellar, brain-stem lesions, and multiple sclerosis.

Exaggerated vertigo is suggestive of a temporoparietal lesion or arachnoiditis; motor-cortex, midline-cerebral, and vermis lesions.

Exaggerated past-pointing is suggestive of occipital-lobe, pituitary, and vermis lesions; prolonged past-pointing is suggestive of frontal-lobe lesions.

Dr. Ben Shuster found exaggerated nystagmus and vertigo in cases of spinal lateral sclerosis syphilis upon turning tests but normal findings upon caloric tests.

Dr. Julius Winston attached a great deal of significance to a disproportion between nystagmus and vertigo responses upon turning tests in a series of cerebellopontile angle tumors he reported some 15 years ago.

Susceptibility to ear stimulation is a very important finding which differentiates a supratentorial from an infratentorial lesion. In some cases even the mass caloric tests cannot elicit the susceptibility without the rotation tests, much less the minimal caloric tests alone.

In a typical posterior-inferior cerebellar artery occlusion there is only absence of responses from one semicircular canal with turning tests; all other responses are normal with standard vestibular tests according to Jones and Fisher. In a recent circumscribed degenerative labyrinthitis the duration of nystagmus elicited from each horizontal canal when turning tests are used is significant.

In irritative and degenerative labyrinthitis (serous and suppurative) and central nervous system lesions, the proportionate and disproportionate impairment in function as determined by both caloric and turning tests is significant in the differential diagnosis.

Wolfson and Fischer state that the loss of turning-test reactions in the presence of a normal caloric reaction in a syphilitic is pathognomonic of inner-ear syphilis.

Some authors find in the disproportion between the turning and caloric tests proof that the two stimuli, the turning and caloric tests, do not act upon the same place in the central vestibular mechanism.

Normal turning reaction with loss of caloric reaction is found in inner-ear syphilis, in certain cases of serous labyrinthitis, and in skull trauma due to changes in the chemical composition of endolymphatic and perilymphatic fluid.

From a physiological point of view the turning reaction has much more significance, since the turning acts as the only adequate stimulus for the semicircular canals and enables one to stimulate the canals in different planes, thus eliciting horizontal, rotatory, and vertical nystagmus from the horizontal, frontal, or superior, inferior, or posterior canals, according to Dr. Spiegel.

In suspected malingering an imbalance of 25-30 seconds of duration of after-turning or caloric nystagmus is against malingering.

In testing for neurotoxicity of the eighth nerve caused by streptomycin or dihydrostreptomycin sulfate therapy, the turning tests indicate impairment before the caloric tests do.

The turning tests are preferable in testing vestibular function in newborn babies and infants.

In cases of atresia or obstruction of the external auditory canals by exostoses, polyps, or malignant-tumor tissue in which the caloric tests can not be used, the turning tests become very useful in addition to the galvanic tests.

Report of Cases

CASE 1.—A 16-year-old boy in the service of S. F. Gilpin, M.D., Temple University Hospital, complained of double vision, staggering, nervousness, and shakiness of two years' duration. He had two cystic growths removed at the Skin and Cancer Hospital.

Neurological examination disclosed a left external rectus palsy, elevation of left eye—1, and ulnar nerve lesion signs on the right. The impression recorded was progressive retrobulbar lesion or sphenoidal ridge tumor.

Ophthalmoscopic examination revealed a 2 D. choked disc in O. D. and normal fields.

Roentgen examination demonstrated a hyperostosis of the cranium with involvement of the frontal bone, osteomata of the ethmoid and lesser wing of the spenoid, with encroachment of the optic canal to a significant degree.

The Bárány tests suggested a midline or bilateral lesion at the base of the cerebral crura, possibly involving the base of the fourth ventricle. Mutiple lesions also involving the cerebral hemispheres could not be ruled out.

The encephalogram was negative.

One year later, in 1948, the patient joined the Army but was discharged two weeks later and hospitalized at the Walter Reed Army Medical Center for two months because of double vision and zigzag walking.

Seven years later the patient was readmitted to Dr. Gilpin's service with the following complaints: wasting of right arm and left leg, diminished hearing bilaterally (past four years in the right ear and past eight months in the left ear), humming in the right ear and bell ringing in the left ear, difficult vision in the right eye, failure to comprehend things well, weakness in the legs, and dropping of the ankles.

Neurological examination at this time disclosed marked atrophy of the muscles of the right arm and left leg, wide base stepping gait, backward falling in Romberg position, motor weakness in all extremities (mostly in the right arm), bilateral foot drop, absence of vibration sense in the left ankle, intact position, and paralysis of gaze of left eye.

Diagnosis: neurofibromatosis, von Recklinghausen's disease.

The Bárány-test findings at this time were indicative of a fourth-ventricle lesion secondary to a cerebellopontile-angle lesion on the right and possibly on the left.

Comparison with the findings of 1947 disclosed marked progressive degenerative changes in the vestibulocochlear mechanism. Nodules in many sites—frontal, parietal, and occipital lobes—revealed a generalized neurofibromatosis upon x-ray and biopsy.

Figure 1.—It took three minutes to elicit poor vestibular function even upon mass caloric tests. The minimal caloric tests probably would not have been adequate to elicit any function. The mass caloric tests alone at this time suggest a midline or bilateral supratentorial lesion and possibly increased intracranial pressure.

Figure 2.—The caloric and the turning tests at this time suggested a cerebellopontile-angle and fourth-ventricle involvement and indicated the progression of the degeneration of the vestibular mechanism.

CASE 2.—A 27-year-old woman was in the service of Dr. Ersner.

Her chief complaints were dizziness and headache of six weeks' duration. Three weeks prior to this attack she had chills and fever, tired easily, felt weak, had cardiac palpitation and shortness of breath, and lost 26 lb. in weight.

Her present recurrent illness dates back six years, at which time she had a severe attack of rotational vertigo and light nauseated her and made her vomit. She was admitted to a local hospital and treated there for an acute suppurative otitis media, which cleared up, and the labyrinthine symptoms subsided.

During the past five years she has had several exacerbations of the chronic suppurative otitis media accompanied by vertigo. Three years ago she had a mastoidectomy on the right mastoid and two years ago a secondary mastoidectomy on the same side for recurrent attacks. After the mastoidectomies while aural discharge persisted she got along fairly well, but as soon as the aural discharge stopped she became dizzy, was nauseated, and had headaches, chills, fever, palpitation, and dyspnea.

At the time of the first mastoidectomy she was considered a cerebellar abscess suspect.

At the time of her third admission to the Temple University Hospital, on Dr. Ersner's service, she had been free from symptoms of sepsis during the past six weeks but her vertigo persisted.

Complete physical and neurological examination failed to disclose any abnormal findings. X-ray of the skull demonstrated a sclerosing left mastoid

Dizziness: none. Tinnitus: yes. Deafness: none. Staggering: yes.

SPONTANEOUS PHENOMENA

Nystagmus
Looking right←few
Looking left→few
Looking up: occasional ↑?
Looking down } none
Looking ahead }
External rectus palsy

Romberg: swaying more to left
Turning head to right: left
Turning head to left: left
Pelvic girdle movements: left

Past-Pointing
Right arm: 1″ to R-T
Left arm: 2″ to L

TURNING TESTS

Nystagmus	Vertigo	Falling	Past-pointing
To right→	12 sec.	To right: none	3″ to R-2″ to R
Duration: 3-4 sec.			2″ to R-1″ to R
Amp.: med., med. frequency			1″ to R-T T
To left←	16 sec.	To left: none	T 1″ to L
Duration: 14 sec.			2″ to R—1″ to L
Amp.: med., med. frequency			2″ to R—1″ to R

CALORIC TESTS

Nausea and perspiration
Right ear — 2″ to R-3″ to R
Verticals ↷
After 40 sec.
Small, slow, inconstant (right eye mostly), improved after 3 min.
Head back: med.,—slow, poor — 3″ to R—2″ to R
Left ear
Verticals ↶ — T T
After 3 min., inconstant, few
Amp.: med., med. frequency — 6″ to L-6″ to L
Head back←
Amp.: med., slow, frequency
Ears:-A.D.-A.C.>B.C.<N.
A.S.-A.C.>B.C.>N. Weber lateralized to the left

Fig. 1 (Case 1).—Testing on July 19, 1947.

Dizziness: none. Tinnitus: humming in right, distortion of sound in left ear.
Deafness: in right ear 3 years' duration, in left ear 6-8 months' duration, subtotal.
Staggering: zigzagging.

SPONTANEOUS PHENOMENA

Nystagmus
Looking right←mod.
Looking left→mod.
Looking up ↑ few inconstant
Looking down ↷ rt. eye
Looking ahead: none

Romberg: falls to left and backward

Past-Pointing
Right arm: 2″ to L
Left arm: can not raise arm

TURNING TESTS

Nystagmus	Vertigo	Falling	Past-Pointing	
To right: none	0 sec.	To right: none	Right arm	Left arm
Duration			T	can not
Amplitude			2″ to R	raise arm
To left: none		To left: none		
Duration	0 sec.		2″ to L	
Amplitude			2″ to L	
			2″ to L	

CALORIC TESTS

Hot
Right ear
Verticals left eye ↶ right eye spont. ↷
After 2 min.
Head back: none except spont. ↷

Cold
Left ear
Verticals left eye ↶ right eye spont. ↷
After 2½ min. variable
Amp.: med., med. to slow frequency
Head back: few with right eye ←
Amp.: med., slow frequency
Ears: A.D.-A.C. none? B.C.-O Weber lateralized to left
A.S.A.C.>B.C.<N.

Fig. 2 (Case 1).—Testing on Nov. 26, 1954.

and the operative defect on the right side (the actively diseased ear).

Hearing was impaired bilaterally, more in the right ear, mixed and obstructive in type. The Bárány-test findings were suggestive of a toxic bilateral degenerative labyrinthitis, rather than a cerebellar lesion.

The right mastoid was revised once more. A contracted mastoid cavity was found with middle fossa and cerebellar dura exposed, which appeared normal. The oval and round windows were easily visualized. A small amount of granulation tissue was removed from mastoid and middle ear. An opening was drilled in the promontory and four to five drops of 95% alcohol were injected into the perilymphatic space.

On the first day following operation the vertigo was more marked. There was a mild spontaneous horizontal nystagmus, first degree, to the right (the operative ear) and a marked spontaneous horizontal nystagmus to the left upon left lateral gaze. Upon upward gaze there was a definite spontaneous vertical nystagmus upward, followed by a rotatory nystagmus to the left.

Eight days postoperatively the spontaneous vertigo and nystagmus had completely subsided, and three days later the patient left the hospital symptom-free. Nine months later the patient was still free from her vertigo generally and was practically well except for a kidney ailment. The right labyrinth was totally destroyed, as evidenced by the vestibular findings.

Dizziness: yes. Tinnitus: right. Deafness: right. Staggering: at times.

SPONTANEOUS PHENOMENA

			Past-Pointing	
Nystagmus			Right arm	Left arm
Looking right ↶		Romberg: falls backward and to R	2" to R	4" to L
Looking left →		Turning head to R: back and to R		
Looking up: none		Turning head to L: back and to L		
Looking down: none		Pelvic girdle movements: unsteady		
Looking ahead: none				

TURNING TESTS

Nystagmus	Vertigo		Past-Pointing	
To right →	10 sec.		8" to R	6" to L
Duration: 12 sec.			4" to R	4" to L
Amp.: med., med. frequency			8" to R	6" to L
To left ←			8" to R	T
Duration: 15 sec.	8 sec.		6" to R	8" to L
Amp.: med., med. frequency			6" to R	8" to L

CALORIC TESTS

Pallor, perspiration, vomiting				
Right ear			2" to R	2" to L
Verticals ↷	Seconds			
After 25 sec.				
Amp.: med., med. frequency			2" to L	T
Head back →				
Amp.: med., med. frequency				
Left ear				
Verticals ↶				
After 90 sec.	Seconds		12" to L	8" to R
Amp.: small, med. frequency		Vomiting		
Head back ←			12" to L	12" to L
Amp.: med., med., to rapid (fibrillary and rapid) frequency				
Bilateral obstructive partial deafness more on right, mixed				
Ears:-A.D.-C.S.O.M. (right mastoidectomy)				
A.S.-C.S.O.M. dry				

Fig. 3 (Case 2).—Testing on Sept. 24, 1943.

Dizziness: none. Tinnitus: none. Deafness: right. Staggering: none.

SPONTANEOUS PHENOMENA

			Past-Pointing	
Nystagmus			Right arm	Left arm
Looking right ↷ mild		Romberg: slight swaying, more to right		
Looking left ↶ mild		Turning head to right: swaying back and to right	T	T
Looking up: none		Turning head to left: swaying forward and to left		
Looking down: none		Pelvic girdle movements: slight unsteadiness to left		
Looking ahead: none				

TURNING TESTS

Slight pallor				
Nystagmus	Vertigo	Falling to right, very mild	Past-Pointing	
To right →	8 sec.		2" to L	2" to L
Duration: 4 sec.			T	T
Amp.: med., med. frequency			T	2" to L
To left ←		Falling to left, very mild	T	T
Duration: 6 sec.	12 sec.		T	T
Amp.: med., med. frequency			T	T

CALORIC TESTS

Right ear		
Verticals: oec. ↷		
After 5 min.	Seconds	
Amp.: very small		
Head back: none		
Amp.		
Left ear		
Verticals ↶	Seconds	
After 90 sec., improved after 2½ min.		
Amp.: small-med., slow-med. frequency		
Head back ←		
Amp.: med., med.-rapid frequency		
Ears:-A.D. Radical mastoidectomy well healed-deaf. B. C. dim; Weber to L		
A.S. Large perforation, dry. Hearing fairly good. B. C. louder		

Fig. 4 (Case 2).—Testing on June 13, 1944.

Figure 3.—Here, again, it took 90 seconds to elicit responses from the left labyrinth, including vomiting, which finding could have been easily overlooked by using the minimal caloric tests. Again, here the turning-test findings reassure one in making the diagnosis of a degenerative labyrinthitis.

Figure 4 is even more convincing as far as the importance of doing mass caloric tests is concerned. Here the right labyrinth is dead as a result of alcohol injection, which is confirmed by at least five minutes of mass caloric stimulation.

CASE 3.—A 50-year-old woman in the service of Dr. G. G. Gibson, Temple University Hospital, exhibited poor vision of 10 years' duration. She is a congenital deaf-mute. A sister and brother had the same defects, with a history of primary degeneration of the retina (retinitis pigmentosa).

The fields showed peripheral contraction. There were posterior polar cataracts. Fundi could not be visualized. Dr. English found no mental retardation.

Radioactive-iodine tests showed 27% retention, which is within normal limits.

Dr. Willson found no evidence of hypogonadism.

X-ray of skull was negative. There was mild contraction of digits of the right hand and left foot but no polydactylism.

Bárány tests indicated almost total aplastic or degenerative neurolabyrinthitis (bilateral).

Final diagnosis was Lawrence-Moon-Beidle disease.

Figure 5.—This case again illustrates the need of adequate stimulation by mass caloric rather than minimal caloric tests plus the turning tests for the reassuring and corroborative additional information.

CASE 4.—A 28-year-old man, referred by Dr. Gilpin, was admitted with the following complaints: He had a stumbling gait for the past two months. For one year he missed occasional steps and his legs were raised too high when he walked, he was unable to run, and there was numbness in his left arm. Six to seven years ago he had blurring of vision, which had increased during the past two months.

Neurologic examination disclosed a questionable scanning speech, ataxic wide base, and high-steppage type of walk. Finger-to-nose test impaired (on left more than on right); marked bilateral ataxia during knee-to-heel test; moderate adiadokokinesis. Atrophy of thenar eminences. Dissociation of lateral gaze; spontaneous horizontal and vertical nystagmus. Weakness of upward gaze. Pallor of optic disc. Decreased vibration sense in legs. Decreased deep pain sense in legs. Babinski's reflex was present on right; abdominal reflexes were absent.

Spinal fluid: Protein content was 240 and 206 mg. per 100 cc.

Ophthalmoscopic examination revealed healed optic neuritis and small central scotoma.

The Bárány tests suggested intracranial lesions—supratentorial and brain-stem lesions.

The findings were not compatible with cerebellar and cerebellopontile-angle lesions.

Final diagnosis was multiple sclerosis.

Figure 6.—Here, again, adequate mass caloric stimulation is desired to inhibit and reverse the spontaneous nystagmus. Again the rotation tests

Dizziness: none. Tinnitus: none. Deafness: yes. Staggering: none				
	SPONTANEOUS PHENOMENA			
Nystagmus Looking right ↷ Looking left ← Looking up ← Looking down ↷ Looking ahead oscillat. ← →	Romberg: neg. Turning head to right Turning head to left Pelvic girdle movements	} no effect	Past-Pointing Right arm T	Left arm T
	TURNING TESTS			
Nystagmus To right: none	Vertigo		Past-Pointing T	T
Duration	Seconds		T	T
Amplitude			T	T-2" to L
Nystagmus To left: none			T	2" to L
Duration	Seconds		T	T
Amplitude			T	2" to L
	CALORIC TESTS			
Right ear Verticals: none			T	T
After 5 min.	Seconds		T	2"-R-T
Amp. Head Back → ? few			T	T
Left ear	Seconds		T	T-2"-L
Verticals: none After 5 min. Amp. Head back: none Amp.				
Ears:—A.D. A.S.	} stone deaf			

Fig. 5 (Case 3).—Testing on Jan. 29, 1954.

Dizziness: none. Tinnitus: buzzing in left ear. Deafness: left impairment. Staggering: stumbling.

SPONTANEOUS PHENOMENA

Nystagmus			Past-Pointing Right arm	Left arm
Looking right ← mild		Romberg: falls to right with eyes closed		
Looking left → mild				
Looking up ↑ mod.		Turning head to right: falls backward and left	1" to R-T	2" to R
Looking down ↓ mild				
Looking ahead none		Turning head to left: falls backward and right		
		Pelvic girdle movements: falls to left		

TURNING TESTS

Nystagmus	Vertigo	Falling to right mod	Past-Pointing	
To right →				
Duration: 20 sec.	24 sec.		6" to R	2" to R
Amp.: med., slow frequency			Vomiting	
To left ←		Falling to left mod.		
Duration: 60 sec.			1" to L	2" to L
Amp.: med., slow frequency	20 sec.		3" to L	2" ro L
Right eye is more active		Vomiting	T	2" to R

CALORIC TESTS

Nausea, perspiration		
Right ear		
Verticals spont. ↓ inhibited somewhat	2" to R	2" to R
after 3 min. no reversal		
after 6 min.	4" to R	T
Amp.		
Head back →		
Amp.: med., med. frequency, slow to start		
Left ear	2" to L	2" to L
Verticals spont. ↓ not reversed after 3 min.		
Amp.: med., slow, inconstant	2" to R	T
Head back ←		
Amp.: med., med. to rapid, frequency, slow to start		
Ears:—A.D.A.C.>B.C.<N.		
A.S.A.C.>B.C.<N. Weber no lateralisation		

Fig. 6 (Case 4).—Testing on Sept. 15, 1954.

exhibit past-pointing within normal limits and, above all, marked susceptibility to ear stimulation, which findings rule out a cerebellar hemispheric lesion in the great majority of cases. The markedly exaggerated nystagmus elicited from the left horizontal is very suggestive of anterior brain-stem irritation and/or multiple sclerosis.

Case 5.—A 47-year-old woman, in the service of Dr. Murtagh, was admitted to Temple University Hospital with the following history: Four years ago the patient began to complain of generalized headache (progressively increasing in intensity) and unsteadiness in gait. In 1952 she had a craniotomy but, due to some complication, the operation was not completed. She improved for about six months, and then symptoms began to recur. She had double vision in all fields and ataxia. Hearing in the left ear decreased until only noise was heard.

Neurological examination revealed diminished corneal reflex on the left, weakness of right external rectus, nystagmus to left, low facial weakness, and hypotonia on left side. DTR reflexes were diminished on the left. There was a Babinski reaction on the left and a bilateral Hoffmann; also dysmetria and dyssynergia to gross and full movements.

Ophthalmoscopic examination by Dr. Sutliff revealed an elevated disc with pallor and a field defect in the left eye due to a lesion anterior to chiasm, most likely papilledema.

Provisional diagnosis by Dr. Murtagh: "The neurological picture certainly incriminates the left cerebellopontile angle, probably brain stem lesion. It is difficult to evaluate the picture since the patient was operated on previously and one can not tell how much is due to postoperative trauma and how much is due to an expanding lesion. A supratentorial lesion should also be looked for."

The Bárány-test findings were indicative of a posterior-fossa lesion with brain-stem involvement rather than an acoustic neurinoma, since there was a moderate amount of bone conduction present and a lesser amount of air conduction and no lateralization of sound.

The ventriculogram demonstrated definitely a large acoustic neurinoma, which was removed by Dr. Murtagh.

Figure 7.—Here, again, the mass caloric tests were preferable to the minimal tests, and the rotation tests demonstrated marked involvement by markedly decreased responses and absence of susceptibility to ear stimulation.

Case 6.—A 58-year-old woman was admitted to Temple University Hospital in the service of Dr. Scott with the following history: Headache and dizziness during the past year. Headaches were intermittent and came and went suddenly. Buzzing in the head, nausea, vomiting, staggering, and pulsating, throbbing tinnitus in head.

X-rays of head and chest were negative.

Neurological examination: coarse nystagmus on the left lateral gaze and fine nystagmus on the right lateral gaze. Eyeground showed slight blurring on the right disc; left disc normal. DTR were slightly increased on the left; bilateral hy-

Dizziness: none. Tinnitus: none. Deafness: left ear, partial, mixed. Staggering: 1 year's duration and weakness in left upper and lower extremities.

SPONTANEOUS PHENOMENA

Nystagmus	Romberg: falls to left and backward	Past-Pointing Right arm	Left arm
Looking right ← rapid			
Looking left → med.			
Looking up ↑	Turning head to right } not tested	4" to R	3" to L
Looking down ↓	Turning head to left }		Tremor
Looking ahead ← few	Pelvic girdle movements		Weakness

TURNING TESTS

Nystagmus	Vertigo	Falling	Past-Pointing	
To right: none			5" to R	8" to L
Duration:	10 sec.	to right: none	6" to R	6" to L
Amplitude			5" to R	8" to L
To left ←				
Duration: 15 sec.	2 sec.	to left: none	8" to R	8" to L
Amplitude: med., med. frequency			5" to R	8" to L
			5" to R	8" to L

CALORIC TESTS

Right ear		
Verticals spont. ↓ rev. to ↷	4" to R	8" to L
After 80 sec.	6" to R	6" to L
Amp.: med., med. frequency		
Head back → spont. ↓	8" to R	6" to L
Amp.: med., med. frequency		
Cold and hot		
Left ear		
Verticals: none		
After 5 min.		
Amp.		
Head back: none		
Amp.		

Ears:—A.D.A.C.>B.C.-N. Weber not lateralized
A.S.A.C.<B.C.<N.

Fig. 7 (Case 5).—Testing in June, 1955.

potonia was present (worse on the left); marked dysmetria on the left with slight ataxia. Marked impairment of pronation, supination, and fine finger movements on the left, no rebound phenomena, left patellar reflex pendular. Walks with a wide base gait and falls to the right.

Diagnosis: left cerebellar leison.

The Bárány tests indicated an intracranial lesion involving brain stem, likely midline posterior fossa. An acoustic neurinoma can be ruled out.

The patient died suddenly during the night.

The pathological report upon autopsy was astrocytoma, Grade 3, midline cerebellar constricting fourth ventricle. Hematoma of cerebellum, cave type of deformity of cerebellum, possible hydrocephalus, acute congestion of spleen, fatty infiltration, severe autolysis and/or necrosis.

Figure 8.—The moderately exaggerated nystagmus revealed by the rotation tests in addition to the inconstant spontaneous vertical nystagmus

Dizziness: 5 months' duration. Tinnitus: in entire head, pulsating, throbbing. Deafness: none. Staggering: to left and forward 5 months' duration.

SPONTANEOUS PHENOMENA

Nystagmus	Romberg: cannot stand; falls backward	Past-Pointing Right arm	Left arm
Looking right ← small			
Looking left → coarse			
Looking up none	Turning head to right } not tested	T	T
Looking down ↓ inconstant	Turning head to left }		
Looking ahead none	Pelvic girdle movements: falls to either side		

TURNING TESTS

Nystagmus	Vertigo	Falling	Past-Pointing	
To right →		Falling to right	6" to R	6" to L
Duration: 35 sec.	10 sec.	mild	4" to R	4" to L
Amp.: med., rapid frequency			4" to R	4" to L
To left ←				
Duration: 38 sec.	10 sec.	Falling to left	8" to R	8" to L
Amp.: med., rapid frequency	Vomiting		8" to R	8" to L
		mild	6" to R	6" to L

CALORIC TESTS

Right ear		
Verticals spont. ↓ rev. to ↷	3" to R	4" to L
After 55 sec., improved after 1½ min.	3" to R	1" to L
Amp.: med., med. frequency		
Head back →		
Amp.: med., med. frequency	T	2" to L
Left ear	4" to L	2" to L
Verticals ↶		
After 20 sec.		
Amp.: med., med. frequency		
Head back ←		
Amp.: med., rapid frequency		

Ears:—A.D.A.C.>B.C.<N. Weber-no lateralization
A.S.A.C.>B.C.<N.

Fig. 8 (Case 6).—Testing on March 26, 1954.

downward suggested the brain-stem involvement, which fitted with the other posterior-fossa findings, such as the spontaneous ataxia in the Romberg position, the markedly diminished duration of vertigo, mild falling, and outward past-pointing. The vomiting which occurred during the turning tests was disregarded in this case, because the patient kept on vomiting before the turning tests were done and the other findings favored more a diagnosis of a posterior-fossa lesion, midline.

CASE 7.—A 50-year-old woman was admitted to the Albert Einstein Medical Center on Medical B. Service with the following complaints: numbness in right upper lip, radiating along right side of nose and right forehead (worse outdoors), of three to four months' duration; right eye was tearing.

She had been dizzy during the last two weeks, had lost her hearing in the right ear, and had developed a sizzling noise bilaterally, worse in the right ear.

Dr. Moore's diagnosis was a right cerebellopontile-angle lesion.

X-ray of skull was negative. Ophthalmoscopic examination was negative. The Bárány-test findings indicated a cerebellopontile angle lesion pattern on the right.

Operation by Dr. Michael Scott revealed a meningioma of the right cerebellopontile angle.

Figure 9.—The history of dizziness, tinnitus, and deafness of two weeks' duration only, the pathological rotatory nystagmus, and the nonresponsive labyrinth after six minutes of mass caloric stimulation were still not sufficient to make a diagnosis of a cerebellopontile-angle lesion without the knowledge of fifth-nerve involvement on the right and the additional information supplied by the rotation tests, which pointed to the fourth-ventricle involvement, namely, the marked depressed responses of nystagmus, vertigo, falling, and the inward past-pointing and the lack of vegetative nervous system symptoms.

CASE 8.—A 41-year-old woman, in the service of Dr. Silverstein, complained of progressive deafness (right) of two years' duration. Ringing in right ear. Attacks of dizziness, numbness of right side of face, headache, visual disturbance, double vision, vomiting spell, burning on right side of tongue, pain in right lower occipital and temporal area, numbness and stinging of right upper and lower lips.

Examinaton revealed the followng: Choked disc, corneal reflex diminished on the right, hypalgesia of fifth nerve, gait with head inclined, swing of left arm, coarse nystagmus to the right, right seventh nerve lower facial weakness. Complete deafness on right; cerebellar signs normal except falling to the right.

The Bárány tests indicated a right acoustic neurinoma.

Eye examination: corneal hypalgesia, o.d. Fields negative, chronic choked disc, nystagmus coarse, vertical.

Dizziness: 2 weeks' duration; 5 years ago imbalance lasted 1 week. Tinnitus: sizzling bilaterally, more in the right. Deafness: right.
Staggering: none at present.

SPONTANEOUS PHENOMENA

Nystagmus	Romberg: slight swaying to right	Past-Pointing	
Looking right ↶ med.	Turning head to right: right	Right arm	Left arm
Looking left ↶ med.	Turning head to left: right	T	T
Looking up ↷ mild	Pelvic girdle movements: right		
Looking down ↶ few			
Looking ahead: none			

TURNING TESTS

Nystagmus	Vertigo	Falling to right	Past-Pointing	
To right →			T	2" to R
Duration: 12 sec.	4 sec.	mild	4" to L	2" to R
Amp.: med., med. frequency			4" to L	2" to R
To left ←		Falling to left		
Duration: 2 sec.	4 sec.		T	2" to L
Amp.: med., med. frequency		none	4" to L	6" to L
			2" to L	2" to L

CALORIC TESTS

Right ear	T	4" to R
Verticals spontaneous ↶ not rev.	2" to L	6" to R
After 6 min.		
Amp.		
Head back: none		
Amp.	2" to R	2" to L
Left ear Hot		
Verticals ↷, later ↶	2" to R	2" to L
After 35 sec.		
Amp.: med. poor		
Head back →		
Amp.: med., med. frequency		

Ears:—A.D. A.C.<B.C.<N. Weber lateralized to left
A.S. A.C.>B.C.=N.

Fig. 9 (Case 7).—Testing on March 18, 1953.

Operation by Dr. Scott: Removal of a large amount of ventricular fluid, also from angle and subarachnoid space above the tumor. An adequate

removal of an acoustic neurinoma was done. Capsule was firmly adherent to the pons and 9th, 10th, and 11th cranial nerves.

Four years later the acoustic neurinoma recurred and was removed again.

Figure 10.—Here, again, the mass caloric tests and the turning tests were more convincing than the minimal tests alone would have been.

CASE 9.—A 52-year-old man was admitted to Temple University Hospital in the service of Dr. Gilpin with the following complaints: intermittent headaches in temples of three weeks' duration and pain in eyes during headaches. He was in usual good health until three weeks ago, when he contracted a cold and began to have pains in back of his head and in the temples and frontal region, which became progressively worse. He had a submucous resection and an antral lavage, but the

Dizziness: yes. Tinnitus: right ear. Deafness: right ear. Staggering: to right, mild.

SPONTANEOUS PHENOMENA

Nystagmus
Looking right ← med. to coarse
Looking left → med.
Looking up ↶
Looking down ↶ coarse
Looking ahead: none

Romberg: slight swaying
Turning head to right } no effect
Turning head to left }
Pelvic girdle movements: good

Past-Pointing: Right arm 1″ to R-T; Left arm 1″ to R-T

TURNING TESTS

Nystagmus	Vertigo	Falling	Past-Pointing	
To right →		Falling to right: very mild	T	2″ to R
Duration: 14 sec.	5 sec.		T	T
Amp.: med., med. frequency			T	T
To left ←		Falling to left: none	T	2″ to L
Duration: 12 sec.	0 sec.		T	T
Amp.: med., med. frequency			T	T

CALORIC TESTS

Cold			
Right ear		1″ to R	2″ to R
Verticals spontaneous ↶ not reversed	Seconds	2″ to L	T
After 6 min.			
Amp.			
Head back → spont.			
Amp.: med., med. frequency			
Hot			
Left ear		1″ to R	1″ to R
Verticals ↶ spont. not reversed	Seconds	T	T
After 4 min.			
Amp.			
Head back → spont. frequency			
Amp. med., med. frequency			

Ears:—A.D. Stone deaf; Weber not heard
A.S. A.C. > B.C. = N.

Fig. 10 (Case 8).—Testing on Nov. 19, 1947.

Dizziness: none. Tinnitus: none. Deafness: none. Staggering: none.

SPONTANEOUS PHENOMENA

Nystagmus
Looking right, Looking left, Looking up, Looking down, Looking ahead } none

Romberg: neg.
Turning head to right } no effect
Turning head to left }
Pelvic girdle movements: good

Past-Pointing: Right arm 4″ to L; Left arm T T

Pelvic girdle movements: good T T

TURNING TESTS

Pallor, perspiration, nausea Nystagmus	Vertigo	Falling to right	Past-Pointing	
To right →			6″ to R	6″ to R
Duration: 28 sec.	10 sec.	mild	1″ to R	4″ to R
Amp.: med., med. frequency			4″ to R	T
To left ←		Falling to left	2″ to R	T
Duration: 32 sec.	20 sec.		8″ to L	8″ to L
Amp.: med., med. frequency		mild	5″ to L	4″ to L
			4″ to L	2″ to L
			T	T

CALORIC TESTS

Right ear	1″ to R	T
Verticals ↶	T	T
After 35 sec.		
Amp.: med., med. frequency		
Head back →		
Amp.: med., med. frequency		
Left ear	4″ to L	4″ to L
Verticals ↶	6″ to L	6″ to L
After 2[illegible] sec.		
Amp.: med., [illegible]ed. frequency		
Head back ←		
Amp.: med., [illegible] frequency		

Ears:—A.D. } no record
A.S. }

Fig. 11 (Case 9).—Testing on Feb. 23, 1943.

headaches became much worse in the suboccipital area, described as hammer blows. One week later a papilledema was found.

Upon neurological examination all the tendon reflexes were subdued and the left patellar reflex was slightly pendular. Left Achilles was less prompt than right. Mild dysmetria on the left. The impression was left posterior-fossa lesion.

Ophthalmoscopic examination: bilateral choked disc.

The Bárány-test findings suggested a supratentorial lesion because the turning tests indicated normal past-pointing and there was moderate susceptibility to ear stimulation.

X-ray of skull was negative.

The ventriculogram, however, showed a space-taking lesion in the occipital lobe on the right.

Operation by Dr. Wycis demonstrated a cyst the size of a walnut in the region of the posterior horn of the right ventricle.

Figure 11.—In this case the turning tests eliminated a posterior-fossa lesion by the fact that the past-pointing was normal and because there was susceptibility to ear stimulation with the turning tests only.

Summary

The standard mass caloric and turning tests are most reliable and informative in neurotologic diagnosis.

The minimal caloric tests are valuable in screening a large number of persons but do not provide an adequate stimulus in cases of moderate or moderately severe impairment of vestibular function. They do not supply the information necessary to make a complete neurotologic diagnosis in the majority of cases.

They are useless in cases of third-degree nystagmus or congenital nystagmus, blindness, amblyopia, albinism, or in reversal of spontaneous nystagmus.

The margin of error is greater with the minimal caloric tests in the various types of pneumatization of the petrons, condition of blood vessels, and psychic states, such as anxiety, fatigue, and excitement.

There is no standard minimal test; the information obtained is inaccurate very often, and the tests are tedious, particularly in cases of moderate or moderately severe vestibular function impairment.

With the mass caloric tests one can employ a minimal threshold stimulus as well as a maximal stimulus for five minutes when needed. The physiologic pessimum and optimum positions are maintained without difficulty. A moderate flow of the irrigating water reaches the posterior-superior canal wall and entire tympanic membrane, obviating the necessity of irrigating the ear with a cannula under direct vision. The 68 F temperature of the water is a proper temperature to test for minimal physiologic deviation from the normal and does not blot out the responses as compared with that of a lower water temperature, such as ice water.

The turning tests supply additional valuable information and should not be omitted from a complete vestibular examination if at all possible.

Dr. Pauline Glanzberg Rachlis assisted in preparing this presentation. Drs. Ersner, Gibson, Gilpin, Murtagh, Scott, Silverstein, and Wycis granted me the privilege of reporting their cases.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

The Cochleopalpebral Reflex in Normal Hearing and Hard of Hearing Persons

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ROBERT A. BUTLER, Ph.D., Washington, D. C.
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In an earlier report, Galambos, Rosenberg, and Glorig [1] investigated the usefulness of the cochleopalpebral, or eyeblink reflex, as a test of hearing. Normal hearing subjects, in addition to patients with hearing loss, were tested to determine the intensity of sound necessary to elicit an eyeblink response. Clicks were used as the auditory stimuli. The authors indicated that the eyeblink response might serve as a measure of loudness recruitment, since some subjects with relatively severe hearing loss demonstrated a cochleopalpebral reflex at sound intensities of only 20 to 30 db. greater than those required for evoking the eyeblink response from persons with normal hearing. This finding suggests that the cochleopalpebral reflex can be used as an aid in differentially diagnosing the type of hearing loss. More specifically, conductively deafened patients should require more sound intensity to produce an eyeblink than most patients with a perceptive-type hearing loss, since there is no experimental evidence that the former diagnostic group exhibits recruitment. In addition, the objectivity inherent in measuring the threshold for the eyeblink response contributes greatly to the value of this technique as a diagnostic tool. The purpose of the present experiment was to examine more thoroughly the cochleopalpebral reflex in normal hearing and hard of hearing persons. Pure tones, rather than clicks, were used as the auditory stimuli.

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Director (Major Albrite) and Research Psychologist (Psychoacoustics) (Dr. Butler), Audiology and Speech Center, Walter Reed Army Medical Center.

Method

Subjects.—In the first phase of this study the cochleopalpebral reflex was investigated, using normal hearing subjects and patients with a perceptive-type hearing loss. Forty persons participated in the experiment. Twenty-one had normal hearing and nineteen demonstrated a perceptive hearing loss. The diagnosis of type of hearing loss was based on complete otologic and audiologic examinations. Within the perceptively deafened group the hearing loss for 10 subjects was attributed to acoustic trauma, 1 subject received a hearing loss following concussion, and 1 suffered a hearing impairment from drug therapy. The etiology underlying hearing defects for the remaining seven patients was unknown.

Apparatus.—The apparatus provided a means of presenting brief loud sounds to the subject and recording the cochleopalpebral reflex elicited by these stimuli. Figure 1 is a block diagram of the equipment. The pure tones were initiated by an audio osciliator (Hewlett-Packard, 200 BR) which was monitored by a Ballantine meter as its output was fed to an electronic switch (Grason-Stadler, 829-A). The electronic switch regulated the rise-fall time of the tones as well as the duration. The input from a pulse generator (Grass Stimulator, Model S4A) "closed" the electronic switch at fixed temporal intervals. The signal transmitted from the electronic switch was fed through an attenuator (Hewlett-Packard, 350A) to a power amplifier (MacIn-

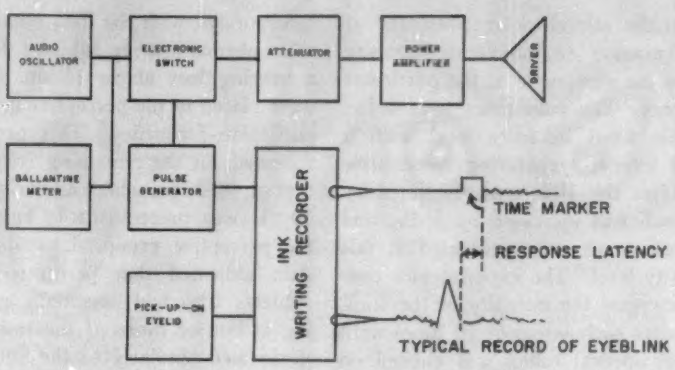

Fig. 1.—Block diagram of the equipment.

tosh, 50W2) which, in turn, actuated a receiver (University Driver, Model PA-30).

The technique of measuring the cochleopalpebral reflex was similar to that described by Galambos and associates.[1] Briefly, one end of a metal pin rested lightly against the eyelid of the subject, and the other end was inserted into an astatic cartridge. The entire assembly was fixed to a headband. By means of a universal joint, located between the cartridge assembly and headband, the metal strip could be placed against the eyelid of all subjects. Eyelid activity produced motion of the metal pin, which caused voltage changes in the cartridge. These changes in voltage were delivered to a Grass Instrument electroencephalograph (Model III-D) for amplification and subsequent recording of eyelid movement. The onset of the stimulus was also recorded. This was accomplished by feeding a signal from the pulse generator directly to the electroencephalograph. The cochleopalpebral reflex could be differentiated from spontaneous eyelid movement on the basis of response latencies to the stimuli. These latencies had a modal value of approximately 45 milliseconds.

The intensity of the stimuli necessary for eliciting the cochleopalpebral reflex was measured for the 500, 1000, 2000, and 4000 cps frequencies. Each stimulus had a duration of 98 milliseconds and a rise-fall time of approximately 8 milliseconds. Because of the size of the receiver required for high intensity stimulation, the earphone was fixed to a vertical wooden frame. The subject was seated in a swivel chair which could be adjusted in height so that his ear was located at the height of the receiver. The patient rested his head so that the nontested ear was fitted to an earmuff located on another wooden frame at a height parallel to that of the receiver. Subjects were instructed to look straight ahead and refrain from blinking their eyes excessively. The continuous noise from the amplifier at high intensities necessitated a spatial separation of approximately 7 cm. between the tested ear and the receiver. Since the hearing acuity for all subjects was nearly the same for both ears, it appeared reasonable to assume that the cochleopalpebral reflex was being initiated by the ear being tested.

The order of presentation for the test frequencies was 1000, 500, 2000, and 4000 cps. Only one frequency was presented daily. With no attenuation in the circuit, the intensity levels *re* 0.0002 dynes/cm.^{2} were 129, 133, 150, and 142 (db.) for the frequencies 500, 1000, 2000, and 4000 cps, respectively, as measured by a sound-level meter (H. H. Scott, Model 410-A). Preliminary investigation indicated that the intensities sufficient to produce an eyeblink response were highly variable for some subjects when the temporal intervals between stimuli were irregular. To avoid excessive variability, all stimuli were given at regular recurring intervals. At the beginning of the

test session the stimuli were presented at 50 db. attenuation *re* maximum intensity provided by the equipment at the particular test frequency. Ten pure tones were delivered at this fixed intensity level with a five-second interval separating consecutive stimuli. After the 10th tone the intensity of the stimuli was increased by 5 db., and 10 additional tones were presented at this new intensity level. The experimenter continued to increase the intensity of the stimuli 5 db. after each series of 10 tones until the cochleopalpebral reflex was elicited on 5 out of the 10 tones presented at a specific intensity. This intensity level was defined as the threshold for the eyeblink response, and testing was discontinued for the frequency. Repeated tests on several subjects showed that the criterion, five eyeblink responses out of 10 tones, appeared to be a stable indicator of the intensity which elicited the reflex phenomenon.

Immediately before and after the test, absolute auditory thresholds were measured by the standard audiometric technique for the test frequency and those frequencies one-half and one octave higher than the test frequency. Obtaining thresholds for the frequency one-half octave above 500 cps was not possible with the audiometer available for threshold measurements.

Results

Some subjects in the normal group had a mild hearing loss at 4000 cps, whereas other subjects in the perceptive group demonstrated normal hearing for the low frequencies and an abrupt drop in hearing acuity for the higher frequencies. To maintain consistency in the analysis of the data, the subjects were classified as having either normal hearing or a perceptive hearing loss with respect to each of the test frequencies. For example, anyone with a hearing loss of 15 db. or less for 500 cps was considered as having normal hearing for this frequency regardless of his hearing acuity for the other frequencies. The threshold data for the eyeblink response to a 500-cps tone were incorporated with the data obtained for the other normal hearing subjects. Patients with a hearing loss above 15 db. for 500 cps were placed in the perceptive group for this particular frequency. This procedure was followed for the remaining frequencies, except at 4000 cps, where a hearing loss above 30 db. was prerequisite to being placed in the perceptive group. Examination of the data indicated that perceptively deafened subjects who had essentially normal hearing at one or more of the test frequencies performed similarly to the subjects in the normal group at these respective frequencies.

The results can be categorized with respect to similarities and differences in performance exhibited by the normal and perceptively deafened groups. Irrespective of the discrepancies between absolute auditory thresholds for the two groups, the intensities required to reach eyeblink response thresholds for the various frequencies appeared to follow a similar function. This finding is graphically illustrated in Figure 2. The two lower curves represent median absolute auditory thresholds, and the two upper curves represent median eyeblink response thresholds. By the method of least squares, the equation $y=0.24x+100$ describes the intensity-frequency function for the eyeblink response threshold in the normal group. The intensity-frequency function for the eyeblink response threshold for the perceptive group can be described by the equation $y=0.21x+107.6$. Only the "y" intercept for the two equations differs appreciably. The most apparent difference between the two groups, however, is the sensation levels for eyeblink response thresholds. The median sensation levels of tones eliciting the cochleopalpebral reflex were 105.5, 107.5, 118, and 120.25 db. in the normal group and 82.5, 72.7, 70, and 70.5 db. in the perceptive group for the frequencies 500, 1000, 2000, and 4000 cps, respectively. There was also less variability associated with the median intensity required to reach eyeblink response threshold

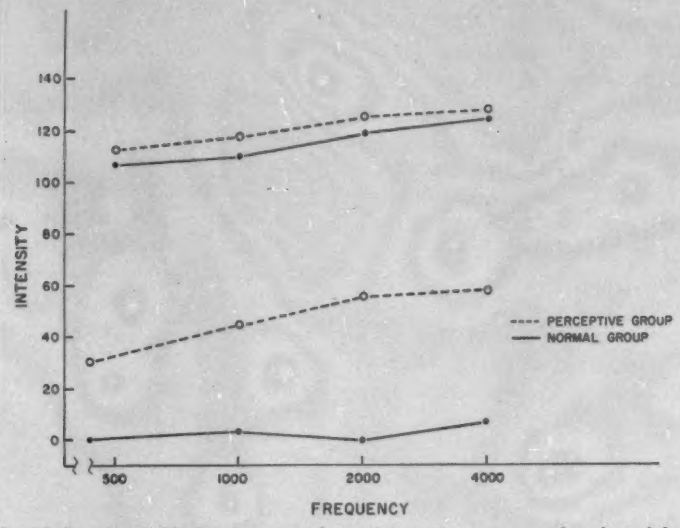

Fig. 2.—Median thresholds for the perceptive and normal groups as a function of frequency.

for normal hearing subjects than for the perceptively deafened subjects. Semi-interquartile ranges for the eyeblink response thresholds for the test frequencies 500, 1000, 2000, and 4000 cps, respectively, were 3.75, 6.5, 4.75, and 7.5 db. in the normal group and 7.0, 6.5, 7.0, and 10.25 db. in the perceptive group. The greater variability in the intensities required to reach eyeblink response thresholds for the perceptive group was probably related to the fact that this group of subjects contained a wide range of hearing losses with different etiologies. For every test frequency, however, the variability associated with median eyeblink response thresholds for the perceptive group was less than that found for the absolute auditory thresholds.

Although the thresholds for the eyeblink response for the normal and perceptive groups were surprisingly similar, significant differences between the normal and perceptive groups were found for the intensity of sound required to elicit an eyeblink response. Table 1 lists the frequency of normal hearing subjects and the frequency of patients with a perceptive hearing loss who demonstrated the cochleopalpebral reflex at various intensities of tones. A x-square test based on the classification shown in Table 1 indicated that the perceptive group required significantly greater intensity ($P<0.02$) to reach the eyeblink response threshold at 500,

TABLE 1.—*Number of Normal Hearing Subjects and Patients with Perceptive Hearing Loss Whose Eyeblink Response Threshold Corresponded to the Various Intensity Categories at Frequencies 500, 1000, 2000, and 4000 Cps*

Eyeblink Response Threshold in Decibels Re 0.0002 Dynes/Cm.²	Normal	Perceptive
	500 Cps	
114-129+	7	12
79-109	19	5
	1000 Cps	
118-133+	8	17
83-113	15	4
	2000 Cps	
125-150+	7	19
100-130	12	4
	4000 Cps	
132-142+	5	12
107-127	14	12

Note: A plus (+) sign indicates that more than maximum intensity provided by the equipment was needed to elicit a cochleopalpebral reflex.

1000, and 2000 cps. No differences between groups with respect to the intensity of tones that elicit the eyeblink response were found for the 4000 cps frequency.

There was no consistent finding for the perceptive group, indicating that the intensity of tone for eyeblink response threshold was directly dependent upon the amount of hearing loss. For example, rank-order correlation coefficients between absolute auditory thresholds and eyeblink response thresholds were 0.07, 0.13, 0.68, and 0.23 for the 500, 1000, 2000, and 4000 cps frequencies. Rank-order correlation coefficients between absolute auditory thresholds and eyeblink response thresholds were not computed for the normal group because of the restricted range of intensities associated with these threshold measurements.

Relatively high intensity stimuli were employed in this study; however, no threshold shifts were obtained in the vast majority of cases. Occasionally there was a 10 and sometimes 15 db. increase in absolute auditory thresholds, particularly for the 6000-cps tone following elicitation of the eyeblink response to a 4000-cps tone. Within approximately five minutes the absolute auditory thresholds returned to the values obtained prior to stimulation. The short duration of the test frequencies, no doubt, prevented large temporary threshold shifts from occurring.

Assuming that the cochleopalpebral reflex is elicited by the loudness attribute of the tonal stimuli, these data suggest that patients in the perceptive group were recruiting loudness. Twenty additional patients diagnosed as having a conductive-type hearing loss were tested in the exact manner first described in order to find out whether this testing technique can be used to differentiate between types of hearing loss. The conductively deafened patients were diagnosed on the basis of complete otologic and audiologic examinations. All patients within this group had hearing losses exceeding 15 db. at 500, 1000, and 2000 cps, and exceeding 30 db. at 4000 cps. The hearing impairment of six of the patients was caused by chronic otitis media. Six other patients had otosclerosis, and the etiology of the remaining eight patients was not known.

Notwithstanding the high intensity stimulation used in this study, most of the patients in the conductive group failed to demonstrate the cochleopalpebral reflex at any of the test frequencies. Moreover, the stimuli did not produce a temporary threshold shift in these patients. When the data for the conductive patients are compared with those for the perceptive patients, it is apparent that the two groups can be differentiated on the basis of the eyeblink response test. Table 2 provides an analysis of the data with respect to type of hearing loss and intensity of tones required to reach the eyeblink response threshold. A chi-square test based upon the obtained frequency of cases in each category listed in Table 2 indicated that differences in performance between the perceptive and conductive groups were significant ($P<0.001$)

TABLE 2.—*Number of Patients with Conductive and Perceptive Hearing Loss Whose Eyeblink Response Threshold Corresponded to the Various Intensity Categories at Frequencies 500, 1000, 2000, and 4000 Cps*

Eyeblink Response Threshold in Decibels Re 0.00002 Dynes/Cm.²	Conductive	Perceptive
	500 Cps	
129+	16	3
99-124	4	14
	1000 Cps	
133+	16	4
108-128	4	17
	2000 Cps	
140-150+	17	5
115-135	3	18
	4000 Cps	
142+	16	6
112-137	4	18

Note: A plus (+) sign indicates that more than maximum intensity provided by the equipment was needed to elicit a cochleopalpebral reflex.

for each test frequency. It is also interesting to observe the remarkable separation between normal hearing and conductively deafened subjects afforded by the eyeblink response test (Table 3). Although the

TABLE 3.—*Number of Normal Hearing Subjects and Patients with Conductive Hearing Loss Whose Eyeblink Response Threshold Corresponded to the Various Intensity Categories at Frequencies 500, 1000, 2000, and 4000 Cps*

Eyeblink Response Threshold in Decibels Re 0.00002 Dynes/Cm.²	Normal	Conductive
	500 Cps	
119-129+	2	19
94-114	24	1
	1000 Cps	
123-133+	1	19
93-118	22	1
	2000 Cps	
135-150+	1	18
100-130	18	2
	4000 Cps	
142+	3	16
107-137	16	4

Note: A plus (+) sign indicates that more than maximum intensity provided by the equipment was needed to elicit a cochleopalpebral reflex.

sound intensities were not sufficient to produce an eyeblink response in most conductively impaired patients, the obtained data suggest that a person with conductive hearing loss must receive an increased sound intensity which compensates for his hearing defect before he will demonstrate a cochleopalpebral reflex.

The possibility that the stimulus intensity at threshold for the eyeblink response might be related to etiological factors underlying hearing loss was examined. No consistent etiological pattern was found for the conductive patients whose cochleopalpebral reflex could be elicited at stimulus intensities comparable to those sufficient to evoke the reflex in the majority of perceptively deafened patients. In the perceptive group, however, it appeared that patients who had experienced acoustic trauma did not require excessive stimulus intensities to evoke the eyeblink response. For example, only one patient whose hearing loss was attributed to acoustic trauma failed to show the eyeblink response at maximum intensities provided by the equipment, thereby performing similarly to the majority of patients with a conductive-type hearing loss. Furthermore, perceptively deafened patients whose hearing loss was caused by factors other than acoustic trauma usually required more sound intensity to evoke the eyeblink response than did those with acoustic trauma.

Comment

The results of this study show that the cochleopalpebral reflex can be used as an aid for differentially diagnosing type of hearing loss. In addition, both the perceptive and conductive groups can be distinguished from normal hearing subjects on the basis of this test. It was tentatively suggested that the sensation level at threshold for the eyeblink response is an indirect measure of loudness recruitment. Whether this phenomenon is actually a manifestation of recruitment in perceptively deafened patients awaits experimental evidence. One approach to this problem would be to correlate the results from alternate loudness balancing measurements with measurements of sensation levels at which the cochleopalpebral reflex is evoked. Hood[2] has suggested that the recruiting ear has normal "on-effect" fibers which provide the illusion of loudness recruitment when the alternate loudness balance test is employed. The technique used to elicit the eyeblink response certainly fulfills the conditions sufficient for activating the "on-effect" fibers. Moreover, the eyeblink response test may be of value in testing for recruitment when other types of tests are not feasible because of the nature of the hearing loss.

Other explanations of the obtained data, aside from a response to loudness, can be postulated. For example, neural impulses from the contraction of the middle-ear

muscles following high intensity stimulation might initiate afferent impulses which activate the eyeblink response. Failure to elicit this response in conductively deafened patients might be attributed to the stiffness of the ossicular chain, which restricts the magnitude of contraction of the middle-ear muscles. However, if the cochleopalpebral reflex, as elicited in this study, is dependent upon contraction of the middle-ear muscles, there would be no apparent reason for differences in performance between the normal hearing and the perceptively deafened groups.

Regardless of the mechanism underlying the cochleopalpebral reflex as evoked by this testing technique, the data clearly indicate that the perceptively deafened group of patients can be differentiated from the group with conductive-type hearing loss on the basis of the intensity of pure tones necessary to elicit the eyeblink response. In addition, both diagnostic groups can be distinguished from the normal hearing group by the eyeblink response test. Whether etiological factors underlying perceptive-type hearing loss might be meaningfully related to threshold intensity for the eyeblink response cannot receive a definite answer on the basis of this study.

Summary and Conclusions

The intensity of sounds necessary to elicit the cochleopalpebral reflex in normal hearing subjects and patients with either a perceptive- or a conductive-type hearing loss was measured. Four pure tones—500, 1000, 2000, and 4000 cps—were used as the auditory stimuli. The presentation of the stimuli and the recording of the eyeblink response were placed under automatic control. The intensity of the stimuli was increased in steps of 5 db. for each group of 10 regularly spaced trials until the subject demonstrated an eyeblink response on 5 out of 10 stimulus presentations at a given intensity level. This intensity level was defined as the threshold for the cochleopalpebral reflex.

The results indicated that the eyeblink response test can differentiate between the normal hearing and the perceptively deafened groups. Moreover, both of these groups can be distinguished from the conductively deafened group on the basis of this test. It is suggested that the cochleopalpebral reflex might be useful as an indirect measure of recruitment, and the objectivity inherent in its measurement contributes to the value of the testing technique.

Pvt. Walter J. Rollin assisted in testing many of the patients and worked on the analysis of the data.

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Clinical Notes, New Instruments and Techniques

A NEW ALCOHOL-CALORIC TEST FOR VESTIBULAR FUNCTION

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While many adequate tests for vestibular, or labyrinthine, function have been described in the literature,[1] we have found the following procedure to be the most satisfactory for our use under most circumstances. Because of its simplicity we feel it worth while reporting for the benefit of those who feel the need for a quick and reliable qualitative determination of labyrinthine function and which can be done with a bare minimum of effort and equipment.

Our method must be considered as similar in results and physiologic response to that described by Kobrak in that it is a "minimal stimulation" test employing an agent to "chill" the vestibular apparatus.

The agent used is a solution of 70% ethyl alcohol, which is a commonly stocked material on almost every physician's treatment cabinet. This is dropped into the external canal of the ear to be tested, with a medicine dropper, after the patient has inclined his head as near to the horizontal as is comfortable for him (Fig. 1). A Boucheron ear speculum of appropriate size is then inserted into the ear canal. With the help of reflected light from a standard headmirror a jet of compressed air, under constant pressure (governed by an ordinary air regulator), is directed toward the posterior-superior quadrant of the tympanic membrane in order to effect cooling as close to the labyrinth as possible (Fig. 2). The air is played on to the surface of the pool of alcohol for a period of 30 seconds, after which the patient's head is held erect and hyperextended upon the neck. His gaze is fixed by having him look at the operator's index finger, which is held some 2 ft. above the patient's face (Fig. 3). By moving his finger toward each extreme of lateral gaze the physician can readily determine the degree of nystagmus

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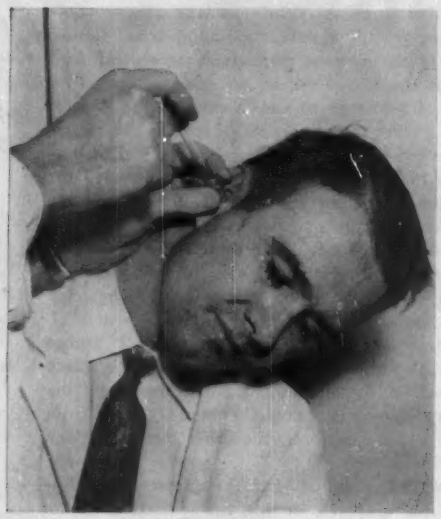

Figure 1

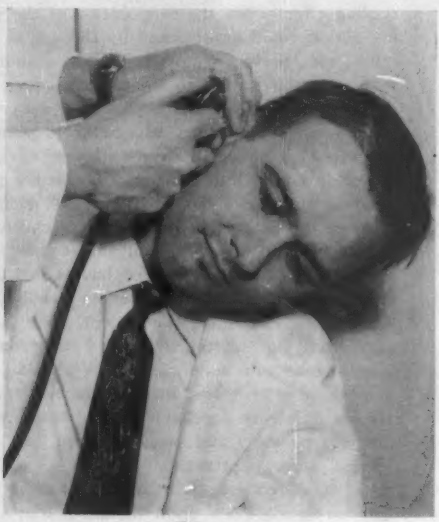

Figure 2

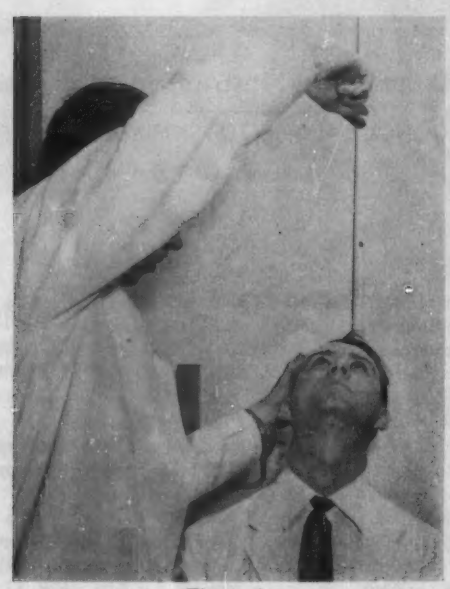

Figure 3

created by the stimulation. An ordinary wrist watch with a sweep second hand on the operator's wrist can serve adequately to time the application of compressed air, the time of onset of the nystagmus, and its duration.

With this method the latent period from the time of onset of the stimulation until the appearance of the nystagmus has been found to average about 40 seconds. The duration of the nystagmus averages approximately 1 minute and 30 seconds. In some instances transient nausea has been noted, but we have yet to note any emesis. A two- or three-minute "resting period" after the nystagmus has ceased has been found adequate prior to testing the opposite labyrinth.

As can well be surmised from the foregoing, the principle of this test is the rapid cooling effect from the evaporation of the alcohol solution. While perhaps greater cooling action could be obtained by the use of more volatile agents, such as acetone, diethyl ether, etc., these are not frequently available in the average physician's office and tend to be quite irritating to the delicate tissues of the ear. Further, we feel that the 70% concentration of alcohol is "safe" and have not hesitated to use it even in the face of middle-ear suppuration, especially in those where the presence of cholesteatoma is suspected. To date, the only adverse effect noted in such instances has been the complaint of a mild burning sensation on the part of the patient. We have felt no need for careful "drying-out" of the ear following the procedure as recommended where the Kobrak test is employed in cases of chronic suppuration.

We refer to this as a "convenient" test for many reasons. First, there is the apparatus employed. There are no ice basins, syringes, irrigating tubing, etc., to be assembled and disassembled. The materials needed are generally within reach of the physician where he is examining the patient. (We have found this to be psychologically beneficial, particularly in children, who tend to become frightened by an array of "gadgets" whose use they do not understand.) While cold-air stimulation alone could be used in cases of perforated tympanic membranes, we feel that such stimulation is not as exact and the results relatively unreliable as compared with those of the alcohol method. Aside from the varying daily and seasonal room and alcohol temperatures, the other test conditions remain quite constant.

It is, of course, quite obvious to the practical otologist that the greatest value of caloric testing rests upon the comparison of the response of one ear with that of the other. It is conceded that the quantitative caloric testing has its place in the university

clinics and should be encouraged when complete neurologic problems are being investigated. In the busy physician's private office the contemplation of such elaborate methods only serves to engender one postponement after another of this vital diagnostic procedure—a deplorable state of affairs for both patient and physician.

While we have not employed this test for other purposes than ascertaining qualitative measure of function of the vestibular apparatus, it is conceivable that this test could be used for more exact neurologic diagnosis by employing the use of such standard neurologic procedures as the Rhomberg sign, past-pointing, etc., during the vertiginous period.

Summary

In the past four years we have performed this simple test in almost every case of systematized vertigo (previously, it must be admitted, the Kobrak test was used only in exceptional cases). Since this alcohol-caloric test can be performed in a very few minutes by the physician himself, if need be, he is sure to renew and revive his interest in the perplexing problems of inner-ear pathology.

Metropolitan Building (2).

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DISPOSABLE RHINOPLASTIC BLADES

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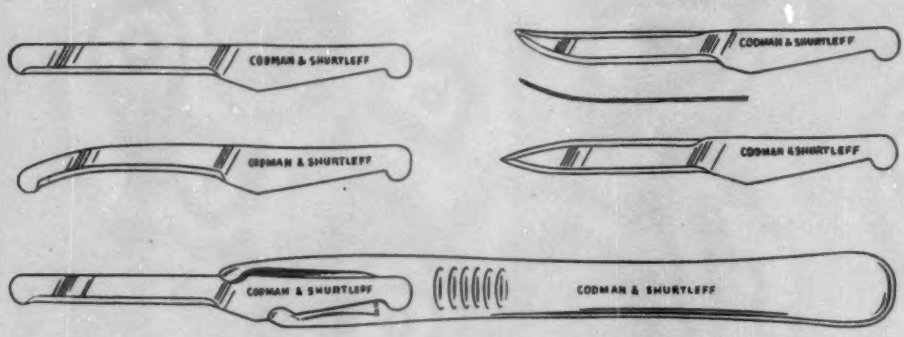
Boston

Numerous sharp knives are the essential tools for all surgery. Straight double-edged pointed knives, double-edged curved blunt-end knives, and button-end straight or curved knives are commonly used. They must be sharp at all times. Sharp blades reduce operative bleeding. They reduce trauma to the soft tissues. They decrease postoperative edema and ecchymosis.

Goldman, in 1952, presented a "throw-away" button-end knife. I have developed a series of blades for use chiefly in rhinoplasty. These are necessary to separate the skin over the nasal pyramid in preparation for reconstruction of the bony and nasal components. They are particularly useful in secondary or revision work.

These blades fit the standard Beaver handle. The blades lock in the handle easily and can be readily removed for replacement by the other blades.

The handles are made of stainless steel. The blades are made of Swedish steel and are 5 mm. wide and 4 cm. long. There are no weakening holes in these blades.



Sketches of Beaver handle and pointed flat double-edged blade, curved blunt-end double-edged blade, straight and curved button-end knives are shown.

Instruments are manufactured by Codman and Shurtleff, Inc., Boston.

REFERENCE

Goldman, I. B.: "Throw-Away" Button-End Knife Blade, A. M. A. Arch. Otolaryng. 55:720 (June) 1952.

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From the Department of Otolaryngology, Massachusetts Memorial Hospitals.

Progress in Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1955

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THE LITERATURE on otitis media and complications for the year 1955 contains an increasing number of articles on intracranial complications. The authors are unanimous in urging careful observations of otitis media cases, early incision of the drum where improvement of symptoms is not rapid, and surgery of the mastoid when necessary. Almost every case of meningitis and brain abscess developed in patients receiving inadequate doses of antibiotics with inadequate supervision.

Antibiotics

Davison [1] (Danville, Pa.) reported on the care of otitis media now as contrasted with its management in the preantibiotic days. In 1937, before the sulfonamides were available, his group did mastoidectomies in 58.9% of 202 cases of otitis media and mastoiditis. In 1943, with the use of the sulfonamide preparations, 26.2% of 282 cases were operated on. In the last few years, with the use of from 1,000,000 to 10,000,000 units of penicillin daily, only 2% or 3% have required mastoidectomy. At the same time deaths from meningitis have been cut down until there have been none in the last three years. He reports no more penicillin reactions with his larger doses than with smaller doses. Although his group has had two anaphylactic reactions to penicillin, they were nonfatal. He is quite certain that, although occasional fatal reactions do occur, many more lives are saved than lost.

He deplored the complacent acceptance of the antibiotics as cure-alls and urged the use of the pneumatic otoscope to determine whether there is fluid in the middle ear.

> In my opinion fluid in the middle ear . . . should always be evacuated by myringotomy and aspiration. . . . It seems pointless to argue whether the use of penicillin is superior to use of myringotomy or vice versa. The sensible attitude is to employ *both* measures as soon as the diagnosis is made. During the last ten years I have not had to perform a mastoidectomy for any patient who came to me for initial treatment of otitis media. . . . The so-called "masking effect" of the antibiotics is no problem for the man who is experienced in the use of these procedures. [Use of pneumatic otoscope, myringotomy, and aspiration.] A normal audiogram is additional evidence of a normal middle ear.

(An excellent paper. The importance of hearing tests should be stressed. With the whispered voice even children as young as 2 years can be tested. Then with a 256 or 512 tuning fork, air conduction is compared with bone conduction [Rinne test]. If the patient hears better by air than bone, he almost certainly does not have pus or serum in the middle ear.)

Silcox [2] (Philadelphia) reported three cases of otitis media with intracranial complications in which the causative organisms were resistant to antibiotics. His first case was a 6-year-old girl with meningitis, lateral sinus thrombosis, and cerebellar abscess. The organisms, beta hemolytic Streptococcus and hemolytic Staphylococcus aureus, were both resistant to penicillin but not to chlortetracycline (Aureomycin). Without extensive surgery she probably would not have recovered. The second case, a boy of 15, had lateral sinus thrombosis and cerebellar abscess. The organisms were hemolytic Streptococcus and Proteus vulgaris, both resistant to all antibiotics. In spite of surgery he died. He was not seen until late;

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earlier surgery might have been successful. The third case was a 68-year-old man admitted to the hospital with meningitis following earache of only three days' duration. The organisms were hemolytic S. aureus and paracolon, both resistant to all antibiotics. A simple mastoidectomy was done, and he recovered.

In the discussion of Silcox's paper, Rosenwasser reported seeing four patients with intracranial complications in the last year and a half. Two had meningitis, one petrositis, and one necrosis of the labyrinthine capsule. All four cases were of two months' or more duration, and all four had dry middle ears. He had found that minimal x-ray findings, relatively normal middle ears, and lack of fever were confusing symptoms. "Mastoid tenderness is still one of the most important signs of bone disease, in spite of the absence of other confirming signs."

Both Davison and Silcox advise penicillin by injection and not by mouth. (There is no question that the clinician can be surer of the absorption when penicillin is given by injection, but several studies have been made showing good blood concentration is achieved when penicillin is given by mouth in large doses.)

McMahon[3] (St. Louis) expressed the following view:

> In our anxiety to accomplish a cure of an infection as rapidly as possible by the early administration of antibiotics, we may lose sight of the fact that it is the resistance of the individual, the action of the immune bodies, the phagocytes, which will eventually cure the patient. . . . It is a lamentable tragedy to deprive a patient of the benefit of an antibiotic in a subsequent serious illness because he has been sensitized at the time of a simple infection which would have responded to ordinary measures.

Where the antibiotics are really needed he advises early sensitization tests to be sure that the right antibiotic is given.

Goodale and Montgomery[4] (Boston) have recently seen 15 cases of otitis media which were neglected to the point of needing mastoidectomy on admission to the Massachusetts Eye and Ear Infirmary. It is their conclusion that the chief cause for these patients coming to operation is the attitude that otitis media carries no great implication of danger; that merely arresting pain and temperature by antibiotics is all that is needed to obtain a cure. They urge early myringotomy and immediate culture. They give case reports of six patients—five of whom developed intracranial complications, none of whom had myringotomy at onset, and all of whom were treated inadequately.

Nakamura and Naganuma[5] (Tokyo) advanced the idea of giving penicillin by injection into the carotid artery for treatment of acute mastoiditis.

> The common carotid artery and the internal jugular vein on the diseased side of the patient with acute mastoiditis were surgically exposed to facilitate the injection of penicillin and the taking of blood from the jugular vein. The concentration of penicillin in the blood of the carotid artery, internal jugular vein, cubital vein, and ear discharge was simultaneously measured. . . .

Twenty-four patients were treated in this way, and twenty rabbits were treated in the same way after injection of bacteria into the middle ear. They found much higher concentrations of penicillin in the blood stream and in the aural discharge when penicillin was given by this method rather than by the intramuscular route. (One of the reasons given for this method of treatment was economy of penicillin. The study was done in 1948, when penicillin was scarce and expensive in Japan.)

Nakamura and associates[6] also used the method of intracarotid injection of penicillin in seven patients with purulent otogenic meningitis. They were able to get much higher concentrations in the cerebrospinal fluid than by intravenous or intramuscular injections. There were six cures. One patient whose family refused operation died.

Diagnosis

Ojala and Palva[7] (Finland) were interested in Bryan's article suggesting that the presence of large phagocytes in the discharge of otitis media would indicate a severe otitis media, usually with mastoiditis. They carefully studied the cytology of 107

cases of otitis media and also studied the cells found in smears of bronchial, pleural exudates in 86 cases of other diseases.

After careful comparison of the types of cells found with the history of the patient's illness, they came to the conclusion that the quantity and quality of the large phagocytes was of no diagnostic help in the prognosis of otitis media. The types of cells found in the ear secretions resembled that in all other infections studied. Large phagocytes were found constantly after the first few days of any infection, whether bone was involved or not.

Everberg [8] (Denmark) published a very interesting report of 15 cases of severe one-sided deafness, probably due to otitis media with extension to the inner ear. In these 15 cases there was (1) unilateral total deafness, (2) absence of vestibular function on the same side, and (3) x-ray evidence of blurred labyrinth with absence of semicircular canals on the same side. X-ray showed normal semicircular canals on the other side.

In 10 of the cases the x-ray showed an apparently normal cochlea on both sides. In four cases there was blurring of the cochlea of the deaf side with normal cochlea showing on the other. In one case the x-ray was unable to visualize the cochlea on either side. In only 4 of the 15 cases was there a history of otitis, and in only 1 was there a history of nausea and vomiting, possibly indicating labyrinthitis.

He reported 55 other cases with total one-sided deafness without x-ray evidence of damage to the labyrinth. Four of these cases were probably due to mumps. There was a positive history of otitis media obtained in 25 of the 70 cases, and scarring of the drum in the deafened ear in 44 cases.

All his cases were otherwise healthy persons, of 7 to 21 years. He thinks most of his cases could be classified as latent otitis, a low-grade infection probably occurring in early life. (The x-ray reproductions are excellent; this paper should stimulate us to watch for similar cases.)

Yannoulis [9] described a new diagnostic sign indicating early weakness of the facial nerve seen in chronic otitis media. On closing the eyes with the head thrown back, the inner canthus of the eye on the side of the affected ear showed delayed closing, and even remained open. In 30 such cases operated on there were changes in the foramen of the facial nerve.

(The original of this article was unavailable; notes were taken from the abstract in *Excerpta Medica* [Section on Otorhinolaryngology].)

Bacteriology

Friedmann [10] (London) studied experimental otitis media in guinea pigs to determine, if possible, whether the principal infecting bacteria had changed in virulence. He used several hundred guinea pigs, injecting many different types of bacteria through the eardrum. Careful microscopic examinations were done at varying intervals.

He found the streptococci (several strains were used) to be less virulent than the strains used by previous workers, all of whom did their experiments before the era of antibiotics. The animals infected with Pseudomonas pyocyanea and Proteus vulgaris suffered most consistently. Infection frequently caused bony destruction of the mastoid, and sometimes there was cholesteatoma formation. The Pneumococcus frequently caused death from meningitis, but clinical symptoms were less frequent. Two strains of Staphylococcus albus (coagulase-negative) caused no infections. None of the strains of streptococci produced fatal infections; most of them caused little or no suppuration.

Shah [11] (Bombay, India) found tetanus organisms in the aural discharge of 14 cases of tetanus. There were 158 tetanus cases in his hospital in six months. Thirty of them had aural discharge, and 45% had positive cultures. Children were more commonly affected than adults. The mortality was 26%, which was less than the non-otogenous series, which ran 48%. Two cases of otitic tetanus recurred months later. He thought that the patients probably in-

troduced dirt into their discharging ears, producing the tetanus infection. (The original of this paper was unavailable; notes were made from *Excerpta Medica*.)

Intracranial Complications

Courville[12] (neurosurgeon of Los Angeles) reviewed the autopsy material from the Los Angeles General Hospital for the five-year period of 1949 to 1954, studying the otitic intracranial complications. The findings were compared with those of his previous well-known analysis covering the years 1928 to 1933. There were 23 fatal otitic intracranial complications in a series of 9737 autopsies in the last five-year period, while there were 124 fatal cases in 5227 autopsies in the preantibiotic era. This shows a drop in death rate of 90%. Meningitis is still the commonest complication. Not one case of lateral sinus thrombosis was found in the autopsies of the second period. Only four cases of brain abscess were seen, two of them developing in chronic ears during flare-ups from acute respiratory infections. "From this it may still be said that a patient with a chronic otitis sits on a powder keg as far as potential intracranial complications are concerned."

Nielsen,[13] (neurologist of Los Angeles) in the symposium with Dr. Courville, stressed the changing and masking of symptoms of intracranial complications since the advent of antibiotics:

> The clinician was a meticulous observer [before antibiotics] who studied his cases.... He knew what was going on at each stage, and it was the sequence of events that gave him his diagnosis.
>
> Now all this is changed. The general practitioner does not make as careful a study of his case, because he has confidence in the antibiotic. ... He tends to forget that trapped pus does not respond to antibiotics and that surgery is necessary for cure of purulent foci anywhere. . . .
>
> Antibiotics have thus reduced the number of intracranial complications of otitis media, but when trouble appears they have made the diagnosis much more difficult. They have had a destructive influence on careful observations and intensive study. They have brought with them a state of trust and confidence in the drugs which in turn lulls the observer into relative dullness. The psychological effect on the physician has been bad. Let him be alert to the fact that we still have complications hiding under a smog through which he must discern the diagnosis and effect a cure.

In discussing the two papers of this symposium, the late J. MacKenzie Brown observed that "Dr. Courville and Dr. Nielsen published a very valuable report on intracranial complications of otitic disease some 20 years ago. . . . Our profession of otology has gained a great deal from their investigations." (I have heard Dr. Brown refer to this paper many times during clinical conferences.) Anderson (Los Angeles) commented that although there were no cases of sinus thrombosis in the autopsy material seen by Courville the condition is far from rare and that at least three such cases were seen per year at Children's Hospital. Day (Pittsburg) mentioned that any patient with an infected middle ear who complained of vague pain and aching in the head must be watched with suspicion of an intracranial infection. Dolowitz (Salt Lake City) then cited a rare case of intracranial infection in which a large amount of air under the temporal lobe was shown by x-ray. This developed slowly after a modified radical operation, performed during the flare-up of a chronic otitis media. The patient had developed pain behind the eye and definite personality changes a few days after she had been discharged from the hospital. Repeat x-rays showed the air to have entered the lateral ventricle, where it disappeared in two weeks. The patient recovered completely without a second operation. Courville, in closing his discussion, thought there may have been a trap-door perforation of the dura which had allowed a spontaneous pneumocephalus. He had never seen such a case secondary to an infected ear. (These two papers should be studied in the original.)

Bastrup-Madsen and Gregers[14] (Denmark) reported on 30 cases of pneumococcal meningitis, most of which were otogenic. They were surprised that the ear symptoms

were so inconspicuous and otological examination so negative. Therefore for the last three years they have performed mastoidectomy in all cases where no obvious other focus could be found and if antimicrobial treatment did not show improvement in a few days. Their best results with the antibacterial medications were achieved with very large doses of penicillin, 10,000,000 units daily, and also large doses of sulfonamides.

Jones [15] (London) reported a case of Pseudomonas otitic meningitis cured by intrathecal polymyxin. The patient, a 19-year-old woman, had had draining ears since childhood. Polymyxin is irritating, causing pains in the legs, but there were no after-effects following doses of 4 mg. daily for 14 days. A short review of the literature revealed only 36 cases of pyocyaneus meningitis reported. He quoted Bancroft as stating 5 mg. is a safe intrathecal dose, but thought 2 mg. daily probably adequate.

Harpman [16] (Warwick, England) reviewed some 70 cases with intracranial complications of ear and paranasal sinus infections. There were 26 cases of lateral sinus thrombosis, 7 cases of otogenic encephalitis, 26 of brain abscess, etc. He was concerned especially with unusual diagnostic problems, including many that resulted from use and misuse of antibiotics and sulfonamides. His paper is impossible to abstract completely and shoud be read in the original. He has observed spread of sepsis following an operation in which a determined effort was made to wipe away all the clot in a thrombophlebitic lateral sinus. He advised draining the pus and softened thrombus but advised ligation of the jugular vein only in those cases which did not improve after the usual measures. In one case of cholesteatoma he found the dura eroded. The defect was covered with a temporal muscle flap and free skin graft. Two years later the patient developed meningitis a few hours after the onset of an acute otitic media. Examination suggested herniation of the brain into the radical mastoid cavity. He waited until the meningitis had responded to penicillin, turned down a temporal osteoplastic flap, dissected up the cerebral herniation with the aid of diathermy, and placed a graft of fascia lata both extradurally and intradurally across the defect.

He agrees with others that it is no longer wise to wait until a brain abscess has encapsulated itself, but that drainage should be done as soon as the diagnosis is definite.

Stuart, O'Brien, and McNally [17] (Montreal) reported a series of 125 cases of brain abscess verified by operation or autopsy. These were seen during the period 1925-1950. About one-half of the 125 cases were due to otitis media and mastoiditis. Intracerebral abscesses were twice as frequent as the intracerebellar. Twice as many abscesses resulted from chronic infections as from acute infections. Meningitis accompanied or preceded cerebral abscess in 23 of the 105 cases. Of the 61 cases of otitic origin, meningitis was present in 18. Intracranial suppuration should be strongly suspected when papilledema, oculomotor paresis, aphasia, stupor, monoparesis or hemiparesis, or defects of the visual fields are found. Convulsions, generalized or focal, indicate abscess above the tentorium. Rapidly increasing signs of intracranial pressure are often the first signs of cerebellar abscess. In addition, pointing errors and coarse horizontal nystagmus indicate cerebellar lesions. There has been an apparent increase of cerebral venous thrombosis lately, "which may be related to the advances of antibacterial therapy [since] some infections were arrested in intermediate vascular stages which might otherwise have gone on to develop brain abscesses." Symptoms of this cerebral venous thrombosis may alarmingly simulate brain abscess and consist of convulsions of arm or face; hemiparesis; aphasia, if the dominant hemisphere is affected, and signs of meningeal irritation. This cerebral thrombophlebitis may well be the same condition sometimes called pseudo-brain-abscess or nonsuppurative en-

cephalitis. (A very well-written and instructive paper.)

Snitman, Singh, Gladney, and Sofranec [18] (Chicago) reported two cases of otogenic brain abscess; one case was unusual in having the organism Achromobacter, and the second, in that there was apparent recovery for two weeks after a radical mastoidectomy only. Both cases developed after flare-ups of chronic otitis media. Both recovered after early drainage through the mastoid cavity. When pus was found with the Cushing needle, the dural incision was enlarged to admit the little finger. A finger cot drain was left in for seven days in one case and eight days in the other. The patients remained well, with no residual symptoms. The Achromobacter organism was sensitive to chloramphenicol (Chloromycetin). The second case was caused by E. coli and was most sensitive to chloramphenicol.

Littell [19] (Santa Rosa, Calif.) reported the case of a girl of 8 who developed a brain abscess after a flare-up of a chronic mastoiditis. Aspiration was attempted by a neurosurgeon through two burr holes, without success. Five days later she was seen by Littell for the first time and immediate mastoidectomy was done. A small circle of dura, 1 cm. in diameter, was electrocoagulated and iodine tincture applied to prevent spread of infection. An aspirating brain needle found the abscess, and 30 cc. of creamy staphylococcic pus was evacuated. A small polyethylene drainage tube was then introduced into the cavity and left 24 hours. The child recovered and has remained normal for two years. Littell values the advice and services of the neurosurgeon but thinks the otologist need feel at no disadvantage in the search for a brain abscess of otitic origin.

Day [20] (Pittsburg) reported the first case he has seen of true congenital cholesteatoma. He had seen many silent cholesteatomas developing from defects in the drum or Schrapnell's membrane. A 45-year-old woman developed a stuffy feeling in the right ear. The posterior half of the drum looked yellow and distended. There had been no known infection. X-rays showed normal pneumatic mastoids with no necrotic changes. Operation showed cholesteatoma filling the middle ear. Since a large radical cavity in a pneumatic mastoid heals slowly and poorly, he decided to leave the mastoid unopened. Atticotympanotomy was done, incus removed because of partial destruction, cholesteatoma removed, and flaps turned up to line the inner attic wall and back to contact the stapes in hopes of better hearing. The hearing did improve about 10 db., and the cavity appeared normal four months postoperatively, at the time of the report. (It has been said that the average otologist will not see more than one true primary cholesteatoma in his lifetime. This is a rare case.)

Lurie [21] (Boston) reported two very unusual cases of cholesteatoma. His first case, a man of 42, had had a left radical mastoidectomy, with skin graft, at the age of 9. He had been well until five years before, when he developed swelling in the left temple with periods of depression and forgetfulness. Operation revealed a cholesteatoma 5 cm. in diameter extending forward under the zygomatic process, and inward into the middle cranial fossa, having pushed the brain aside for 5 cm. The postauricular incision had to be extended forward to the external angle of the orbit. Eight years later, because of recurring headaches, the cavity was reopened and another layer of cholesteatoma, 2 mm. thick, was scraped off the dura and temporal muscle. It was impossible to remove all the cholesteatoma or to externalize the cavity, so the patient was told that he would probably need another exploration in another 8 or 10 years.

In his second case, cholesteatoma invaded the petrous pyramid and required two operations, the first a Neumann operation, which showed cholesteatoma in the bony vestibule. When he began to have heaviness in the side of the head 11 years later, the cavity was reopened, the cochlea exenterated, the carotid artery and jugular bulb exposed,

and the cholesteatoma was found extending into the petrous apex.

He concluded that cholesteatomas outside the mastoid cavity in the temporal bone are difficult to eradicate; that one should be alert for symptoms of recurrence and ready to reexplore when symptoms recur, even though the cavity may appear clean.

Permin,[22] (Copenhagen) being interested in fistular labyrinthitis, reviewed the histories of all patients with chronic ears admitted to the ear clinic of the Copenhagen City Hospital from 1945 to 1954. There were 856 patients, 108 of them with positive fistula tests. In the 95 patients who responded to follow-up letters, only 25 had positive fistula tests; 51 had dry ears after operation, and 44 had chronic or periodic discharge. Only one-half of the postoperative patients had useful hearing. He concluded that the fistulas caused by erosion of cholesteatomas and infection very frequently heal by bone or heavy scar tissue, so that there is no longer a positive fistula test.

Howie[23] (Glasgow) reports the case of a woman of 36 who developed jugular thrombosis and a deep cervical abscess. She had a chronic otitis of 20 years' duration. She had chills and fever for 15 days and had been treated by a general physician with several antibiotics before consultation was called for. The first operation revealed a large cholesteatoma with exposure of the lateral sinus but no apparent thrombus. On the 10th postoperative day the patient again developed chills and fever. The lateral sinus was inspected at a second operation and a needle puncture produced blood. Palpation showed no clot; therefore the internal jugular vein was exposed and found to be collapsed but not infected; but as it was dissected up to the base of the skull an abscess was found, from which Proteus vulgaris was cultured. Howie observed that undue trust in antibiotics caused delay that may have been the cause of the abscess requiring the second operation.

Weaver[24] (Detroit) reported the unusual case of a woman of 28 who developed a facial paralysis and also paralysis of the spinal accessory nerve as a result of a cholesteatoma of the right mastoid. She had had a draining ear since the age of 2. There was a large subperiosteal abscess, from which was cultured Pseudomonas aeruginosa. Four months after the radical mastoidectomy there was normal motion of the face and shoulder. The reason for the paralysis of the spinal accessory nerve was not clear. There was no similar case found in the literature in the past 10 years.

Cawthorne[25] (London) discussed the surgery and management of intracranial complications due to chronic otitis media. He advised that the intracranial infection be subdued first, then the primary source of infection could be cleared up. He cited one case in which a temporal lobe abscess had been drained and finally excised. Shortly before the patient was to be discharged he again became very ill and was transferred to the otologic service. Immediate operation found a quantity of foul pus between the dura and bone.

He reported two cases of suppurative labyrinthitis, both of which had caused meningitis. In both cases radical mastoidectomy was done but the labyrinth was left untouched. Both cases developed meningitis again, one of them after three months. Both of them recovered after the labyrinth was opened and pus drained. He opens the labyrinth both above and below the facial nerve. He does not hesitate to do this if tests show a dead labyrinth. "To leave a fistula in a bony semicircular canal is to invite future trouble from vertigo." He recently did a labyrinthectomy on a patient that had been bedridden for five years because of vertigo. She was walking in two weeks and working in two months. She had had 17 previous operations on the same ear, without relief of vertigo. Another patient, a man in his middle 30's and a window cleaner by trade, had been totally disabled for two years by persistent vertigo. He had a dry mastoid cavity with an active fistula sign, the other ear being normal.

After a labyrinthectomy and balancing exercises he returned to his window-cleaning work within three months. Cawthorne feels strongly that in dealing with intracranial infections or in chronic ears with very poor hearing a complete radical mastoidectomy should be done. He has seen a number of intracranial complications develop in patients after modified radical operations, but never after a radical. (A very well-written and interesting paper.)

Chronic Mastoiditis

Meltzer [26] (Boston) has given us a very readable summary of his management of the chronic middle ear that requires surgery. He is very insistent in advising the preservation of the mucous membrane where the hearing is at all serviceable, especialy if the hearing in the other ear is not good. He uses primary skin grafts where it is necessary to clean out the middle ear thoroughly, but is sure that hearing is impaired in these cases. "The only time I have observed good hearing in a patient with a skin graft over the medial wall was when, for some unexplained reason, an air space developed under the graft in the region of the windows." He reminds us that the basic principles of radical mastoid surgery were clarified in the last century. His rules of procedure, modified from Richards, and his quotations from Dench and others make the paper well worth reading in the original article.

Dingley [27] (London) relates that he has become more conscious of the importance of conservation of hearing in chronic infections of the ear and advises early modified radical operations before the hearing is too badly damaged. He has been especially interested in the effect of removal of the incus and its later effect on hearing. In the past he felt that hearing was not too much affected by removal of the incus. He now thinks that in some cases the hearing may be good in spite of removal of the incus but that in some cases much loss of hearing resulted. He now examines the incus at operation carefully. If it is glistening, firm, and whole he retains it. He opens the aditus widely, removes adhesions and cholesteatoma. In several cases in which cholesteatoma extended down from the attic into the middle ear, the drum and ossicles were left with resuting dry ears and good hearing.

Secretory Otitis Media

Hitschler [28] (Philadelphia) reported the interesting case of a woman of 40 whose serous otitis was so severe and prolonged that a simple mastoidectomy was finally performed. On opening the mastoid there was pink fibrous tissue in the mastoid antrum and middle ear. There was much yellow serum in the region of the mastoid antrum, but the cells distal to the antrum were normal. The patient was normal after operation, but the hearing loss of 30 db. was not recovered. He thought that if the operation had been done earlier the hearing might have been preserved. Before the operation, frequent myringotomies were first tried, then a small polyethylene tube was left in the myringotomy incision for three weeks. While the tube was in place the ear was comfortable; when it was removed, the ear refilled at once. Thyroid extract was also tried, without improvement, before operation.

External Otitis

Tremble [29] (Montreal) gave a very thorough review of the medications used in otitis externa and agreed with others that there was no one medication that worked best in all cases. He emphasized the importance of an acid reaction of the medication and quoted Ochs, who advised the use of household vinegar. Recently, on a trip to Africa, he found a well-informed doctor there who had tried all the modern medications and preferred commercial vinegar. He used full-strength vinegar, pouring it into the ear canal six or eight times daily. Tremble has been using the cortisone preparations with good results. He strongly urges that the underlying causes, such as the allergic, physical, endocrine, and emotional factors, be corrected or recurrences will occur.

Senturia, Sophian, Chiang, and Lowry [30]

(St. Louis) reviewed a number of experiments this group has been carrying on, with help from the Air Force and others. Microchemical methods of analysis have been worked out to determine the chemical formation of normal cerumen and the secretions in external otitis. They have also studied biopsy specimens in external otitis and found complicated inflammatory changes, including compression and atrophy of the glands in the acute stage and a complete absence of sebaceous glands in the chronic stage. "These observations support the theory that the underlying mechanism in the pathogenesis of diffuse external otitis may be a malfunction of the epidermal glands." As for treatment, one study used five antibiotic preparations, and Senturia was impressed with the lack of superiority of one preparation over another. "In certain chronic otitis cases striking results were obtained with the topical use of various preparations containing hydrocortisone."

Senturia [31] (St. Louis) reported on the continuation of his excellent work on external otitis. He advised removal of the patient from a hot, humid environment and irrigation of the ear canal with hypertonic saline; in addition, for the mild case, use of aluminum acetate or Burrow's solution; for the severer case, oral use of broad-coverage antibiotics. As improvement occurs he advises 10% boric acid in wool fat (anhydrous lanolin ointment) with continued cleansing treatment until normal wax begins to form again. He has found that irrigations with isotonic saline sometimes cause flare-ups but has never seen an exacerbation from the use of 3% hypertonic saline. He prefers the use of aqueous preparations to those using ethylene glycol.

Technique

McAskile [32] (Toronto) described his technique in handling the soft tissues in temporal-bone surgery. His conclusions are based on the care of 1500 cases. He has observed that residents going into practice have had their chief concern from soft-tissue complications, such as stenosed meatuses and granulations under the conchal margin, rather than from improper bone surgery.

For the simple mastoid operation he prefers the postauricular incision for three reasons: (1) more direct access to the mastoid cells; (2) lessened possibility of development of perichondritis; (3) recurrent mastoiditis following the endaural incision may cause bulging into the external canal, making it difficult to differentiate between furunculosis, external otitis, or recurrent mastoiditis. During the last two years he has closed the postauricular incision completely in 6 simple mastoidectomies, in 9 decompressions of the facial nerve for Bell's palsy, and 12 cases of destruction of the labyrinth for hydrops. All these cases have healed by primary intention.

His detailed description of his modifications of the endaural incision will be worthwhile reading for those not satisfied with their results.

He has had four dural tears with spinal-fluid leakage and one sigmoid sinus tear with hemorrhage. All occurred in cases where pathological exposures were present and where an attempt was being made to remove cholesteatomatous matrix. He thinks that the matrix produces a thinning or atrophy in such cases.

He advises skin grafts to cover all raw surfaces. "Careful selection of a line of incision 6 mm. deep to the conchal margin makes it possible to suture the meatal skin to the periosteum, and completely eliminate any exposure of cartilage."

Campbell [33] (Philadelphia) describes his technique of skin grafting radical mastoidectomy cavities and his results in 50 cases. His procedure includes careful removal of all pathology, including the matrix. He uses a Brown electrodermatome and cuts a very thin graft, 3 by 4 in., from the inner surface of the upper arm on the side of the infected ear. The graft is laid on a strip of fine-meshed gauze soaked in isotonic saline. Four strips 1 in. wide are cut and laid in the mastoid cavity so as to cover the

entire area with some overlapping. The packing, which has been soaked with antibiotic ointment, is not removed for 7 to 10 days. He is sure his patients get dry ears sooner with less troublesome after-care.

Fine [34] (Johannesburg, South Africa) compared the healing time of 106 skin-grafted mastoid cavities with that of an ungrafted group of 38. The ungrafted cases required an average of 15.3 weeks, while the grafted series healed in an average of 9.2 weeks.

Myers and Wilf [35] (Philadelphia) describe their modification of the endaural incision. They found that most of the granulations and resulting stenosis were prevented by a flap which included a large area of skin from the cavum conchae, cut with a broad base which is left attached. (Their photographs and diagrams are very clear.)

Farrior and Iglesias [36] (Tampa, Fla.) reported their technique by which they do a primary skin graft in their radical mastoidectomies and fenestrations. When there is postoperative infection they wash the cavity with 1 to 4 fluidounces (30 to 120 cc.) of 1%-2% acetic acid in 70% alcohol and teach the patient to do the same at home. Where long continued care is needed they have the patient use one part vinegar and three parts alcohol rubbing compound. They allow their patients with radical mastoid or fenestration cavities to swim but advise them about the possibility of a caloric reaction to cold water. Filling the canal with cotton is advised to prevent this dizziness. Rinsing the ear afterward with the acetic alcohol mixture seems to prevent any otitis. In the discussion Shambaugh stated that he had given up skin grafting because of increase of "troublesome desquamation, walls of the cavity are more irreguar and harder to clean, and the cavity is larger. With scrupulous asceptic technique in the post-operative care, a great majority of these cavities heal very nicely by themselves with a healthier appearing skin and a smoother surface when the epidermis grows in by itself over a thin pad of granulation tissue."

(The discussion on the merits of skin grafting continues. Howard House is now performing secondary grafts on the 10th day, using a local anesthetic in the outpatient department. This saves a second general anesthetic and the expense of a second admission to the hospital. Dr. Edward Dench, one of the pioneers in mastoid surgery, used to do his skin grafting on the 10th day, and I well remember his excellent results.)

Tympanoplasty

Frenckner [37] (Stockholm) reported 50 cases of tympanoplasties, part of them done by Wullstein's technique of free grafts and part by the author's technique of a pedicle graft. He advises that in most cases, especially if there is cholesteatoma, the modified radical operation should be done first, and the tympanoplasty later.

Wullstein [38] (Wurzburg, Germany) reports the results of tympanoplasties in 200 cases. There was improvement in 53.5%, no change in 43%, and a worse condition in 3.5%. Plastic operations on chronic middle-ear cases are divided into three groups: no affection of ear bones, ear bones affected but not the stapes, total destruction of ossicles. Each of these classes requires a different operative technique. (Article unavailable; abstract taken from *Excerpta Medica.*)

Eosinophilic Granuloma

Hoxie [39] (Jacksonville, Fla.) reported a case of eosinophilic granuloma of both temporal bones in a man of 34. In spite of severe dizziness and other symptoms over a period of 12 years, he had been many times treated by penicillin injections for a two- or three-day period. Microscopic examination of the tissue removed from his mastoid showed eosinophilic granuloma. He later developed foci in the shoulder, jaw, and pubis. X-ray treatment of the mastoids and other affected areas produced an apparent cure. The author thinks it possible that the repeated doses of penicillin may have acted as an allergic factor in causing aggravation and extension of the granuloma to

new sites, since there was renewed activity in the mastoid operated on (left) after the administration of 47,000,000 units of penicillin.

Miscellaneous

Chusid and Bernstein [40] (New York) reported the case of an infant 6 days old to whom iodized oil was given during a gastrointestinal series because of partial obstruction. Skull x-rays were also done, and iodized oil was found in the right middle ear and mastoid. X-rays taken at 2 days of age for possible skull injury had showed no such opacities. Five months later x-rays showed opacities still present but smaller and less dense. They reported this as illustrating a mechanism by which infections of the middle ear might occur.

960 E. Green St.

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Abstracts from Current Literature

Ear

TRANSTYMPANIC MOBILIZATION OF THE STAPES FOR IMPAIRED HEARING DUE TO OTOSCLEROSIS. C. M. Kos, Ann. Otol. Rhin. & Laryng. 64:995 (Dec.) 1955.

After 65 transtympanic stapedial mobilization operations, Kos has learned a number of pertinent facts and has gained some interesting impressions.

The technique is satisfactory, but it is a hit-or-miss procedure, with no detailed criteria evident for selection. Predictions cannot as yet be made with a significant degree of confidence.

According to the author, three categories of clinical otosclerosis must be considered for the stapedial mobilization procedure: (1) incomplete stapedial ankylosis, (2) complete stapedial ankylosis, and (3) stapedial ankylosis with inner-ear hearing deterioration. The first two categories are based on normal bone-conduction thresholds for pure-tone audiometry. Analysis of the cases in this series seems to confirm the observation that best results are to be obtained in cases of beginning ankylosis, the operation not being effective in patients having complete ankylosis of the stapes. However, the "cross-representation of success and failure in the two groups does not significantly clarify the issue of selection and prediction."

While regression of hearing improvement may occur in both Groups 1 and 2, it is found more frequently in the cases of beginning ankylosis. Usually it occurs four to six weeks postoperatively but in the series at hand has occurred as late as six months after operation.

The operation is most effective in the third group, where bone-conduction thresholds are so elevated as to preclude the possibility of a successful fenestration operation. In such cases, the hearing may be restored to the level of the bone-conduction threshold. It is concluded that whether stapedial mobilization will be retained in the surgical repertory of the otologist will depend upon future experience with the procedure and the long-term results to be evaluated in the next several years.

STEELE, Kansas City, Kan.

RECOVERY PATTERN FROM AUDITORY FATIGUE. JAMES F. JERGER, J. Speech & Hearing Disorders 21:39 (March) 1956.

Pattern of recovery of the auditory threshold after intense stimulation is characterized by a secondary maximum or "bounce" at approximately two minutes after cessation of the fatiguing stimulus. With a two-minute fatiguing tone at a frequency of 3000 cps and an interrupted threshold tracing signal of 4000 cps as the SPL of the fatiguing tone was increased from 80 to 95 db., the mean recovery curve showed a gradual increase in the magnitude of the "bounce." Above 95 db., however, a sudden acceleration in initial threshold shift was accompanied by a gradual decrease in the magnitude of the bounce. At an SPL of 110 db. the bounce had virtually disappeared. Disappearance of the bounce from the recovery curve may mark the transition from fatigue to reversible acoustic trauma.

PALMER, Wichita, Kan.

CONDITIONED EYELID RESPONSE TO TONE AS AN OBJECTIVE TEST OF HEARING. THOMAS E. GALLOWAY and ROBERT A. BUTLER, J. Speech & Hearing Disorders 21:47 (March) 1956.

A study of 22 subjects ranging in age from 19 to 52 at the Audiology and Speech Center, Walter Reed Army Medical Center, showed that the conditioned eyelid response can be used in an objective test of hearing. The chief virtue of this technique is the precision with which the conditioned eyelid response can be defined. The differences between thresholds obtained by audiometric tests and those measured by this conditioning technique averaged approximately 5 db.

PALMER, Wichita, Kan.

SEROUS OTITIS MEDIA. J. DUDLEY SINGLETON, Laryngoscope 66:293 (March) 1956.

This paper concerns mainly the diagnosis, treatment, and management of serous otitis.

The cells which line the middle ear and mastoid favor a transudation of fluid in the presence of a vacuum.

Eustachian-tubal closure from any cause is the exciting factor. The onset is sudden. There is a full feeling of the ear (or ears) and hearing loss. Audiophony and the feeling of fluid in the ear are common symptoms. Occasionally there are tinnitus and vertigo. The appearance of the eardrum aids in the diagnosis. Local treatment is indicated. Daily inflation (Politzer method) should be performed several times, followed by myringotomy (sometimes repeated), if necessary. Politzerization and self-inflation should, in turn, follow the myringotomy. Suction is sometimes necessary to evacuate a very thick accumulation.

In the resistant cases, an autogenous vaccine may be used or a semipermanent opening in the drum may be made. Very occasionally a mastoidectomy is required.

HITSCHLER, Philadelphia.

RELATIONSHIP BETWEEN ETIOLOGY OF HEARING LOSS AND RESULTANT AUDIOMETRIC PATTERN. W. W. WILKERSON JR. and JONATHAN I. H. DOYLE, Laryngoscope 66:550 (May) 1956.

One hundred fifty-one cases of hearing loss are analyzed as to (1) audiometric pattern and (2) etiology. Based on the work of Ciocco and Carhart, the cases are divided into eight audiometric patterns, while 10 different etiological groups are considered. In addition to the pure-tone audiometry, each case was tested with speech audiometry. Other allied tests were performed.

The cases falling into each of the eight audiometric patterns are analyzed. They are also analyzed as to the 10 etiological divisions. There were enough cases in several categories for determination of the statistical significance of the observed results. However, this was not true in other groups because of the paucity of the cases. The study of a much larger group of cases would be desirable.

HITSCHLER, Philadelphia.

INDUSTRIAL DEAFNESS. C. STEWART NASH, New York J. Med. 56:1626 (May 15) 1956.

Nash has long been known as one of the key men in the University of Rochester School of Medicine and Dentistry. His contributions to otolaryngology have been numerous and intensive. In this essay he reviews what has been achieved in the studies of deafness among workers. This is one article in a series entitled "Recent Advances in Medicine and Surgery," under the editorship of Robert Turell, M.D. Formerly, manufacturers were rather regardless concerning noises in factories, but they have come to know that "a noisy product meets sales resistance." Noise experienced for hours at a time may bring exhausting fatigue of the whole body plus hearing loss. It is becoming increasingly important that examination before employment must take particular account of aural conditions, past and present, and include all tests for hearing loss and dizziness (from Ménière's disease or other causes). Insurance underwriters have considerable interest in this matter, and compensation bureaus need all the help they can get from any source in making a decision on awards. Everyone concerned needs to know whether a given person is suffering from acoustic trauma (temporary loss) or acoustic degeneration (permanent loss). The former comes on suddenly as a result of violent impact by direct contact or through the air (explosions). Occupational deafness comes on gradually as a result of long-continued exposure to noises, that is, from day to day. The industrial hazard which an employee will be exposed to ought to be made known to him before he signs up for a job. Likewise, employer and insurance underwriter should have in mind their responsibilities for a worker's health and life. It is obtuse to neglect this viewpoint. Acoustic trauma can occur at any place at any time: in one's home, on the street. It is often thought of as temporary, since recovery is almost the rule, but the hearing loss can be permanent. Nerve or perceptive loss of hearing, usually permanent, means a failure of the brain center to perceive the stimulus coming to it. A third type is mixed deafness, in which both the conductive and perceptive functions are reduced or lost. Occupational deafness is classified as an industrial disease.

Several questions must be asked and answered in any litigation or discussion of industrial noise and occupational deafness: Is the noisy environment a contributing cause of partial or complete loss of hearing? The question of degree comes up. Also to be considered are the type of person affected, his tolerance of noise, and whether he had previous trouble with his ears. Ability to construe correctly the spoken word is the basis of all compensation awards. What of the age factor? Allowance for normal aging effect on hearing is set at 0.5% for each year after age 55. Below that age no allowance is made. The Board believes it would be fair to grant a 5% allowance up to 60 years, that is, from 51 to 60. After age 60 the

allowance would be 10%. Otologists who wish to work in the relatively new field of industrial medicine must study, or at least know, what the engineers, physicists, research workers, etc., are doing to reduce damaging noises to the lowest possible level. To that end one may find the "Report of the Committee of Consultants on Occupational Loss of Hearing" of very considerable interest. It may be obtained by writing to the New York State Workmen's Compensation Board, State Office Building, Albany 1, N. Y.

I. W. VOORHEES, New York.

TOXIC ACTION OF STREPTOMYCIN SULFATE ON THE EIGHTH NERVE. GABRIEL CAPELLA, Acta oto-rino-laring. ibero-am. (Barcelona) 7:97, 1956.

In reviewing the literature concerning the toxic effect of streptomycin sulfate and dihydrostreptomycin on the eighth nerve, Capella quotes the various authors who had determined various pathologic processes on variors portions of the eighth-nerve system. To substantiate these findings, he has undertaken to study the effect of streptomycin in rabbits. From his observations he found the following: 1. There is no relationship between the dose administered and either the functional or histologic abnormalities which can occur with streptomycin. He believes that there must be a constitutional factor which is responsible for these disturbances. 2. In all his cases he found a hypoexcitability on rotation. 3. The pathology is usually a peripheral lesion and is more intense in the cresta than in the macula. 4. There is a vacuolization of the sensory cells, with an intense loss of the cilia and gelatinous substances. In the maculae, in some cases, the cilia and the otolytic membrane were retained in appreciable quantities. 5. Finally, he could not find any appreciable lesion in the cochlear or vestibular nuclei.

PERSKY, Philadelphia.

Pharynx

GENERAL TONSILLECTOMY. PAUL M. MOORE, Ann. Otol. Rhin. & Laryng. 64:494 (June) 1955.

Moore presents his methods for tonsillectomy and adenoidectomy in children and adults as follows: With the utilization of isotonic saline containing six minims (0.35 cc.) of 1:1000 epinephrine to the ounce, injections are made into the supratonsillar and infratonsillar areas, anterior and posterior pillars, and peritonsillar area prior to surgery, for the purpose of aiding hemostasis. About 10 cc. of the solution adequately prepares both tonsils for dissection. The adenoid area is injected with the same solution after removal of the adenoid if bleeding persists. Sponge pressure is then used if bleeding still continues. If this fails, a sponge moistened with epinephrine and dusted with tannic-acid powder is applied. This is followed by ligature of any bleeding points, the author preferring the use of the slip-knot ligature to use of suture.

In certain cases, injection of isotonic saline and epinephrine solution is contraindicated. Moore advises against the use of epinephrine in children under the age of 2, in diabetic patients, in adults who have evidence of coronary artery disease, and in any instance when cyclopropane anesthesia is employed.

According to the author, the method of peritonsillar injection outlined above reduces the amount of bleeding during adenotonsillectomy and aids in the dissection.

STEELE, Kansas City, Kan.

Larynx

PROGRESS IN LARYNGOFISSURE. MERCER C. LYNCH, Ann. Otol. Rhin. & Laryng. 64:746 (Sept.) 1955.

Lynch outlines the cases of laryngeal carcinoma in which laryngofissure is the procedure of choice and lists his technique in the operative details, complications, and follow-up.

It is concluded that laryngofissure for carcinoma of the larynx may be approaching a point of development where it is possible "that in the majority of patients with carcinoma of the larynx adequate removal of the tumor can be accomplished without destroying the function of the larynx."

STEELE, Kansas City, Kan.

LARYNGEAL PAPILLOMATA. DEGRAAF WOODMAN, Ann. Otol. Rhin. & Laryng. 64:794 (Sept.) 1955.

Woodman presents a case of persistent multiple papillomata occurring in a woman, aged 35, in which the laryngofissure approach was used to effect removal of papillomata on one side by submucosal resection and on the other side by alar perichondrial elevation, removing both true and false cords from the anterior commissure to the vocal process. Bleeding was controlled by cautery. An acrylic mold was attached to a tracheotomy tube to afford a snug fit. The larynx and neck tissue were closed in routine fashion.

After the acrylic mold was allowed to remain in the larynx for eight weeks, it was removed and the glottis was found to be smooth and covered by normal mucosal lining. Although there was no recurrence at the site of operation, the author has removed papillomata from the trachea on two occasions since the laryngeal surgery.

According to the author, the procedure is of value in those cases which develop obstruction and laryngeal stenosis due to papillomata.

STEELE, Kansas City, Kan.

BENIGN PEDUNCULATED ESOPHAGEAL TUMORS. FRANCIS E. LEJEUNE, Ann. Otol. Rhin. & Laryng. 64:1261 (Dec.) 1955.

The occurrence of a benign pedunculated upper esophageal tumor in a 36-year-old man is reported by LeJeune. After esophagoscopy, which did not permit complete visualization of the tumor, the patient regurgitated the neoplasm into the hypopharynx, obstructing the glottis. Asphyxia, anoxia, and eventual death resulted. The autopsy specimen then demonstrated the true pedunculated nature of the lesion, a fibroma.

Benign tumors of the esophagus are rare. The rarest of all are of the pedunculated variety. They are of two types, intraluminal and extraluminal. Those of the intraluminal variety originate in the mucosa or submucosa and occur usually in the upper portion of the esophagus. They gradually become pedunculated due to gravity pull, swallowing, and laxity of the esophageal mucosa. These tumors are usually adenomas, fibromas, hemangiomas, and lipomas and are frequently encountered first by the esophagoscopist.

The extraluminal tumors arise from the outer wall of the esophagus and cause symptoms by exerting pressure on the esophageal wall. There are more of this type of neoplasm now being reported by those doing thoracic surgery.

Benign esophageal tumor may cause no symptoms, but, when it does, the usual manifestation is progressive dysphagia. Roentgenography and esophagoscopy should establish the diagnosis. Treatment consists in peroral excision, when possible, but cervical esophagotomy is frequently necessary.

STEELE, Kansas City, Kan.

HOARSENESS IN LARYNGEAL PATHOLOGY: A REVIEW OF THE LITERATURE. JOHN M. PALMER, Laryngoscope 66:500 (May) 1956.

Three conditions cause hoarseness: laryngeal carcinoma, paralysis, and tuberculosis. Normal phonation requires that the vocal cords be capable of approximation, tension, and vibration, and any interference of these functions will result in hoarseness. The various factors which may interfere with each of these are discussed. A very thorough examination, not just confined to the larynx, is often necessary to determine the nature of the disease.

Cancer and other growths affecting the larynx are discussed. Hoarseness is the first and most important symptom. The degree of change of the voice is, of course, dependent upon the site and size of the lesion. There may be variations from complete aphonia (some lesions in the anterior commissure) to no voice loss at all (lesions located above the glottis anteriorly.

In laryngeal paralysis, hoarseness is also a common symptom. It varies according to the type of paralysis and may even be absent in unilateral involvement when the healthy cord crosses over the midline to compensate. On the other hand, hoarseness comes on very gradually, has a moist sound, and varies in amount from day to day. There is a gradual weakening of the voice.

HITSCHLER, Philadelphia.

HOARSENESS AND PAINFUL DEGLUTITION DUE TO MASSIVE CERVICAL EXOSTOSES. CHARLES V. HECK, Surg. Gynec. & Obst. 102:657 (June) 1956.

The author presents four cases which demonstrate extreme alterations in the cervical

spine resulting from exostoses due to osteoarthritic changes. Clinically the complaints of hoarseness and difficulty in swallowing may be outstanding. The bronchologist may by pure coincidence be the first to notice the possibility of such a disturbance when instrumentation of the esophagus becomes impossible because of a block.

The onset of hoarseness in these cases has been gradual, and pain in the cervical spine area or radiation to the neck was not a major complaint. Sometimes a firm bony mass may be palpated anteriorly in the affected area. Treatment is difficult and must be planned for each case. It may be necessary to resort to gastrostomy when poor nutrition is a problem. The detailed case reports for patients presenting this problem are given by the author.

FRIEDBERG, Chicago.

Nose

VASOMOTOR RHINITIS. THEODORE E. WALSH, Ann. Otol. Rhin. & Laryng. 64:466 (June) 1955.

Walsh states that he is not able to identify allergic factors in a large percentage of his patients with "vasomotor rhinitis" but that endocrine, dietary, and psychosomatic disturbances play the major role in the etiology.

Objectives of management are (1) to determine and treat the course of the vasomotor state and (2) to provide the patient with some measure of relief during the investigation. The use of local vasoconstrictors is inadvisable in symptomatic treatment. Better relief of nasal congestion can be secured by the use of vasoconstrictors by mouth, such as phenylpropanolamine (Propadrine), with or without antihistaminics.

If the allergic history is suggestive, appropriate skin tests and dietary regulation should be performed. Those patients who show a low basal metabolic rate and high blood cholesterol readings respond well to thyroid medication. Taking vitamins, such as 50 mg. of thiamine hydrochloride and 0.5 gm. of ascorbic acid daily, is helpful.

STEELE, Kansas City, Kan.

OSTEOMA OF FRONTAL ETHMOIDAL SINUS. J. PORTELA DE VILLASANTE, Acta oto-rino-laring. ibero-am. (Barcelona) 7:143, 1956.

The author presents a case of an osteoma of the frontoethmoidal sinus which was operated on with satisfactory results. In presenting this case he also reviews the literature and draws the following conclusions: 1. Most of the osteomas can be extirpated by means of fronto-ethmoidal incision. 2. If the osteoma does not involve the dura or the lamina cribrosa, the extirpation of the tumor by Cushing's method is illogical. 3. If a pyocele or a mucocele is associated with the osteoma, it can be treated most easily by the frontonasal incision. 4. Those osteomas associated with intracranial complications can be treated most easily by a transfrontal incision or by the method of Cushing. 5. Finally, if the osteomas are associated with intracranial complications and a secondary infection of the sinuses, the cure can be effected if surgery is performed in two stages.

PERSKY, Philadelphia.

Miscellaneous

OBSERVATIONS AND CONSIDERATIONS ON CIGARETTE SMOKING. MERVIN C. MYERSON, Ann. Otol. Rhin. & Laryng. 64:412 (June) 1955.

The author has briefly reviewed the recent literature relative to the use of tobacco and its effect on the human body, with particular emphasis on the respiratory tract. He concludes that excessive smoking affects the larynx, pharynx, and tracheobronchial tree. The relationship between cancer of the lung and excessive cigarette smoking, though suggested by statistical studies, has not yet been scientifically established. It is believed that efficient filters and filter tips on cigarettes are of value in reducing irritation caused by smoking.

STEELE, Kansas City, Kan.

PROPHYLAXIS OF RHEUMATIC FEVER. MACLYN MCCARTY, New York J. Med. 56:1670 (May 15) 1956.

This essay was delivered over Station WNYC-FM under the aegis of the New York Academy of Medicine. Dr. McCarty is affiliated with the Hospital of the Rockefeller Insti-

tute for Medical Research. Rheumatic fever is of interest to ENT men because this disease is commonly preceded by streptococcal infection of the upper airways. "There can no longer be any serious doubt about this relationship. . . ." Elimination of streptococci from the throat effectively prevents the occurrence of rheumatic fever. Penicillin is the only antibacterial drug which has proved to be completely reliable. The sulfonamides are ineffective. Penicillin must be given in adequate dosage for at least 10 days, and if there is danger of allergic sensitivity, then oral administration is in order. Throat cultures are imperative, especially the isolation of Group-A Streptococcus. Recurrences of rheumatic fever have been known to be as high as 50% in past years, and with long periods of reminiscences in between attacks. The sulfonamides are said to be of some use in prophylaxis, but leucopenia must be watched for.

ABSTRACTER'S COMMENTS: As far back as 40 years ago I called attention to the *post hoc* evidence of rheumatic fever following "sore throat." However, the thought was not accepted, even by so great a researcher as Dr. Homer Swift, of the Rockefeller Institute. Now that we recognize the sequence, it is the responsibility of every ENT specialist to culture all sore throats and to apply oral sterilization plus penicillin when Streptococcus is found, and sometimes when this enemy is not found.

I. W. VOORHEES, New York.

DISCUSSION ON CARCINOMA OF THE THYROID. I. DONIACH, Proc. Roy. Soc. Med. 49:173 (April) 1956.

The meeting of the Royal Society of Medicine, Section of Surgery, was held Nov. 2, 1955, under the presidency of John Bruce, C.B.E., T.D., F.R.C.S. Ed. Dr. Doniach has been working with the induction of benign and malignant thyroid tumors in rats. It seems that there are regions where rats live which are endemic goiter districts. Adenomas were found commonly and, occasionally, carcinomas. It was determined that iodine deficiency is associated with goitrogenic tumors.

The common factor in these experiments was thyroid hyperplasia, with a raised output of pituitary thyrotrophic hormone (T.S.H.). The sequence was the following: generalized hypertrophy and hyperplasia of the thyroid; the focal development of adenomata in sharply demarcated areas with a morphology distinguishable from that of the surrounding thyroid tissue; malignant change, with increase in the size of the thyroid and permeation of extracapsular veins, often with metastatic deposits in the lungs. In man it is thought that the majority of thyroid carcinomas arise from adenomatous goiters (Rundle, 1951). It may be that excessive T.S.H. stimulation in childhood and adolescence may establish "a legacy of adenomas" which appear later in life after the stimulus to hypertrophy has ceased. From the United States Simpson and associates have reported x-raying of the thyroid in infancy for enlarged thymus or tuberculous lymph nodes of the neck, followed, eventually, by cancer of the thyroid. The role of T.S.H. as inductive to adenomatous human goiters is still sub judice. It is suggested that small doses of thyroxine, sufficient to prevent excessive pituitary secretion of T.S.H., might prove helpful in preventing both adenomas and carcinomas in later life. Some 16 references are given to the literature.

I. W. VOORHEES, New York.

News and Comment

ANNOUNCEMENTS

Sixth International Congress of Otolaryngology.—The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

Chronic Suppuration of the Temporal Bone

Openers: Marcus Diamant, Central County Hospital, Halmstad, Sweden
Anatomical Etiological Factors in Chronic Middle Ear Discharge
Luzius Rüedi, Zürich, Switzerland
Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone
Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany
Surgical Repair for Improvement of Hearing in Chronic Otitis Media

Discussers: A. Tumarkin, Liverpool, England
Juan Manuel Tato, Buenos Aires, Argentina
T. E. Cawthorne, London, England
Fritz Zöllner, Freiburg, Germany

Collagen Disorders of the Respiratory Tract

Openers: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada
Introduction
Michele Arslan, Padua, Italy
The Upper Respiratory Tract
Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.
The Lower Respiratory Tract

Discussers: Victor E. Negus, London, England
Branimir Gusic, Zagreb, Yugoslavia
Aubrey G. Rawlins, San Francisco, U. S. A.
Henry L. Williams, Rochester, Minn., U. S. A.

Papilloma of the Larynx

Openers: Jo Ono, Tokyo, Japan
Etiology
Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil
Diagnosis
F. C. W. Capps, London, England
Therapy

Discussers: C. A. Hamberger, Göteborg, Sweden
Pedro Hernandez Gonzalo, Havana, Cuba
Eelco Huizinga, Groningen, Netherlands
Albert von Riccabona, Vienna, Austria

Applications to present voluntary papers are being received regularly, and anyone wishing to present such a paper should make known his intentions prior to the deadline of Oct. 1, 1956.

The motion-picture sessions, as well as the scientific exhibits, will prove to be outstanding in every respect. It is essential that anyone wishing to present an exhibit should make his intentions known immediately, as the final date for consideration is Aug. 1, 1956; applications to present motion-picture films should be sent in before Oct. 1, 1956. Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

Joint Meeting of the Society of Military Ophthalmologists and the Society of Military Otolaryngologists.—The Society of Military Ophthalmologists and the Society of Military Otolaryngologists will hold a joint stag dinner meeting at the time of the Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, to be held in Chicago in October.

Dinner will be served at 6:30 p.m. on Oct. 16, 1956, at the Palmer House. The dinner will be followed by a short business meeting.

All members of the Society of Military Ophthalmologists and the Society of Military Otolaryngologists are invited to attend. Application may be made to either Capt. James A. Stokes, MC, Secretary-Treasurer, Society of Military Ophthalmologists, Eye Clinic, Walter Reed Army Hospital, Washington 12, D. C., or to Major Stanley H. Bear, MC, 3810th USAF

New Members of Editorial Board.—The Trustees of the American Medical Association, at its June Meeting, elected the following new members to the Editorial Board of the A. M. A. ARCHIVES OF OTOLARYNGOLOGY. Dr. Victor Goodhill, of Los Angeles, was elected to fill the vacancy created by the lamentable death of Dr. John MacKenzie Brown. Dr. George E. Shambaugh Jr., son of Dr. George E. Shambaugh Sr., who was the founder and first Chief Editor of the *Archives of Otolaryngology*, fills a newly created post, concerned largely with the Chicago district.

A Special Postgraduate Course.—An Introduction to Fundamentals of Reconstructive Surgery of the Nasal Septum and External Pyramid will be presented by Yale University School of Medicine, Department of Surgery, Section of Otolaryngology, in cooperation with The American Rhinologic Society and Grace-New Haven Community Hospital May 11-18, 1957.

The course will be given under the guest direction of Dr. Maurice H. Cottle. It will consist of didactic lectures, case presentations, cadaver dissections, and observation of operations. All sessions will be held at the Yale-New Haven Medical Center, New Haven, Connecticut, U. S. A.

Enrollment will be limited.

Applicants must be diplomats of the American Board of Otolaryngology or its equivalent for doctors from other countries. Scholarship assistance is available to a limited number of foreign applicants.

For further information and application forms please write to the Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar St., New Haven 11, Connecticut, U. S. A.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. G. M. T. Hazen, 208 Canada Building, Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.
Place: Banff Springs Hotel. Time: June 17-19, 1957.

International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth International Congress of Bronchoesophagology.
Place: Philadelphia. Time: May 12 and 13, 1957.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Habana.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: 1958.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958 (afternoons only).

American Laryngological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Washington, D. C. Time: May 3, 1957.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy E. Ireland, 100 College St., Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Joseph Sullivan, Toronto, Canada.
Place: Royal York Hotel, Toronto, Canada. Time: Jan. 31-Feb. 1, 1957.
Southern.—Chairman: Dr. Mercer G. Lynch, New Orleans.
Place: Roosevelt Hotel, New Orleans. Time: Jan. 16-17, 1957.
Middle.—Chairman: Dr. Paul M. Moore, Cleveland.
Place: Hotel Cleveland, Cleveland. Time: Jan. 9-10, 1957.
Western.—Chairman: Dr. Sigurd v. Christierson, San Francisco.
Place: Hotel St. Francis, San Francisco. Time: Jan. 19-20, 1957.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37.
Secretary-Treasurer: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Place: Statler Hotel, Washington, D. C. Time: May 4, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

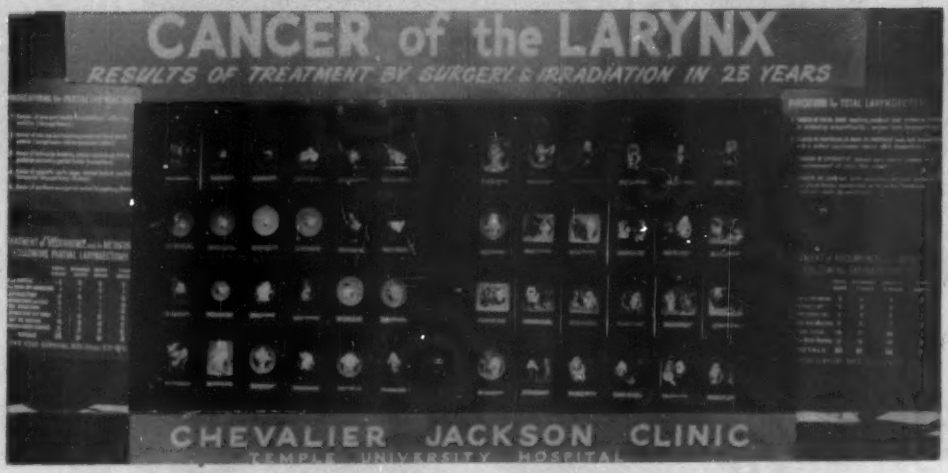
AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Dean McAllister Lierle, University Hospital, Iowa City.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS

President: Col. Wendell A. Weller
Secretary-Treasurer: Major Stanley H. Bear, M. C., 3810th USAF Hospital, Maxwell AFB, Ala.
Place: Palmer House, Chicago.

Scientific Exhibits


CANCER of the LARYNX
RESULTS OF TREATMENT BY SURGERY & IRRADIATION IN 25 YEARS
CHEVALIER JACKSON CLINIC
TEMPLE UNIVERSITY HOSPITAL

CANCER OF THE LARYNX

Study Based on Twenty-Five Years' Experience

CHEVALIER L. JACKSON, M.D.
JOHN V. BLADY, M.D.
and
CHARLES M. NORRIS, M.D.
Philadelphia

The exhibit shows by color transparencies of operative specimens, the varied appearances of carcinoma of the larynx and laryngopharynx. Criteria for the selection of treatment are presented in relation to end-results, and detailed information concerning the treatment of recurrence and metastasis is presented.

Temple University School of Medicine.

PRIMARY TREATMENT IN 1022 CASES of CARCINOMA of the LARYNX (1930-1954 INC)

	PARTIAL * LARYNGECTOMY	LARYNGECTOMY	LARYNGECTOMY SIMULTANEOUS RADICAL NECK	IRRADIATION
1930	7	15	0	0
1931	5	10	0	1
1932	8	8	0	1
1933	14	5	0	4
1934	8	12	0	4
1935	8	12	0	8
1936	7	11	0	9
1937	14	11	0	9
1938	5	5	0	13
1939	12	22	0	7
1940	11	16	0	6
1941	12	9	0	20
1942	6	19	0	14
1943	13	15	0	13
1944	16	12	0	10
1945	16	13	2	13
1946	23	11	0	17
1947	21	23	0	17
1948	16	12	0	24
1949	22	20	0	19
1950	20	21	7	16
1951	25	18	5	12
1952	27	13	9	8
1953	19	22	9	10
1954	23	17	14	11
TOTALS	358	352	46	266

* (Laryngofissure, Partial Horizontal (Alonso), Partial Frontal etc.)

FIVE-YEAR SURVIVALS INCLUDING SALVAGE FOLLOWING RECURRENCE and/or METASTASIS

	DET. CASES	5 YR. SURVIVAL FOLLOWING PRIMARY Rx		RECURRENCE and/or METASTASIS	5 YR. SURVIVAL FOLLOWING FURTHER Rx	TOTAL 5 Yr. SURVIVAL INCLUDING Rx of REC./MET.	
Partial LARYNGECTOMY	208	182	88%	26	8	190	91%
LARYNGECTOMY	206	131	64%	75	7	138	67%
IRRADIATION	179	73	40%	106	11	84	47%
TOTALS	593	386	65%	207	26	412	69%

FIVE YEAR SURVIVAL RATE (412/593) 69%

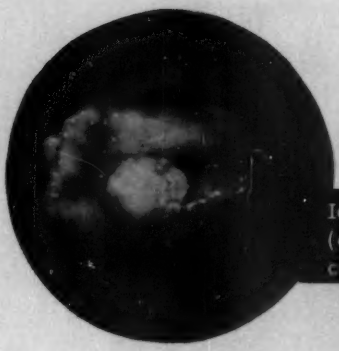
INDICATIONS for PARTIAL LARYNGECTOMY

1. Cancer of one cord limited to middle third, with free motility (laryngofissure)
2. Cancer of one one cord reaching anterior extremity but not posterior. (laryngofissure - anterior commissure technic)
3. Cancer of both cords involving anterior commissure but no posterior extremity (partial frontal laryngectomy)
4. Cancer of epiglottis early cases - limited lesions (partial horizontal laryngectomy - Alonso)
5. Cancer of pyriform sinus (partial vertical laryngectomy - Alonso)

TREATMENT of RECURRENCE and/or METASTASIS FOLLOWING PARTIAL LARYNGECTOMY

	TOTAL CASES	DETERMINATE CASES	DEAD OF DISEASE	5 YEAR SURVIVAL
2nd PARTIAL	2	2	0	2
2nd PARTIAL THEN LARYNGECTOMY	1	1	0	1
LARYNGECTOMY	4	4	1	3
LARYNGECTOMY and X RAY	3	1	1	0
NECK DISSECTION	1	1	1	0
LARYNGEC. X RAY N. D. RADON	1	0	0	0
X RAY OR RADIUM	9	8	6	2
UNTREATED or TREATED ELSEWHERE	4	4	4	0
TOTALS	25	21	13	8

FIVE YEAR SURVIVAL RATE (Salvage) 8/21 = 38 %

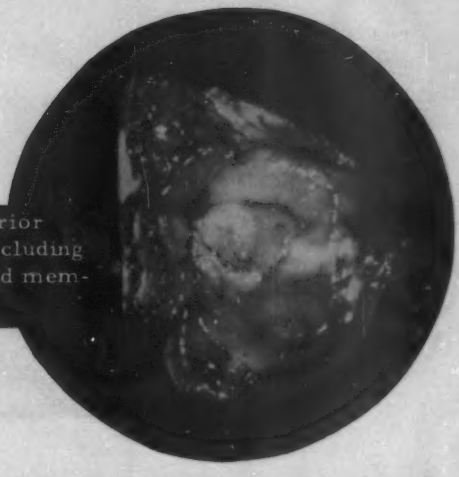


Ideal lesion for treatment by laryngofissure (clipping technic). Specimen includes entire left cord, ventricle and portion of ventricular band.

Lesion of middle third of right vocal cord excised by laryngofissure (clipping technic). Right cord and ventricle excised along with underlying portion of thyroid ala.

Carcinoma of right cord, specimen including cord, ventricle and portion of ventricular band, with underlying thyroid ala. Excision by laryngofissure (Clipping technic).

Lesion of anterior commissure and anterior subglottic region, excised by partial frontal laryngectomy.

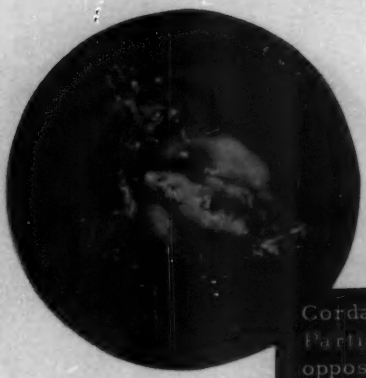


Carcinoma of anterior commissure and anterior half of right cord. Partial laryngectomy, including anterior extremity of left cord, crico-thyroid membrane and most of right thyroid ala.

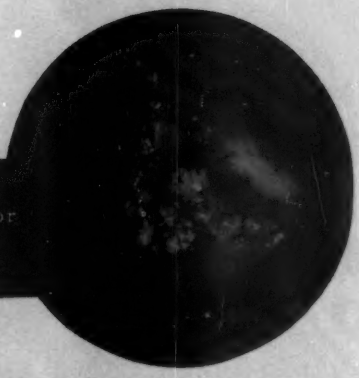
Carcinoma of right cord, with beginning subglottic extension. Partial laryngectomy, including anterior end of left cord and ventricular band and large part of thyroid ala. No post-op. stenosis.

Typical excision of cordal lesion reaching anterior commissure. Partial laryngectomy, including anterior end of opposite cord and ventricular band, plus anterior vertical segment of thyroid cartilage.

Carcinoma of anterior two-thirds of right cord, reaching anterior commissure. Excised specimen includes large portions of arytenoid and crico-thyroid membrane, with anterior third of left cord and band.

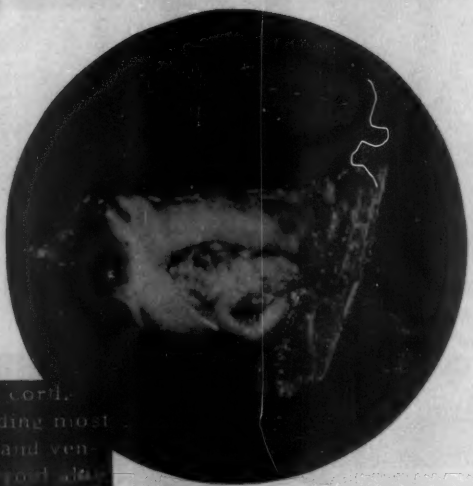


Cordal lesion approaching anterior commissure. Partial laryngectomy, including anterior end of opposite cord and ventricular band with attached anterior portion of thyroid cartilage.



Carcinoma of anterior commissure and right cord. Partial laryngectomy, including anterior portion of left cord and ventricular band and vertical anterior segment of thyroid cartilage.

Infiltrating lesion of right cord, reaching anterior commissure. Partial laryngectomy including anterior half of left cord, underlying cartilage and entire arytenoid. Immediate graft - no stenosis.

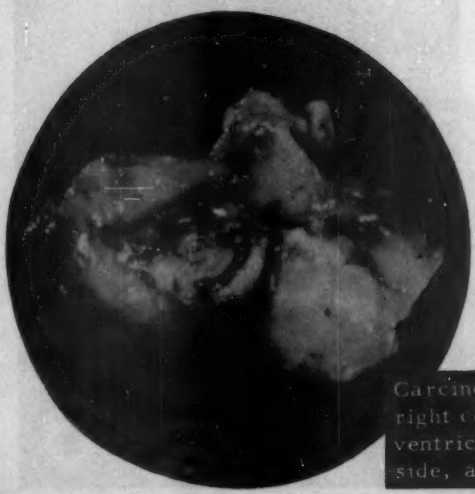


Carcinoma of anterior two-thirds of left cord. Excision by partial laryngectomy, including most of arytenoid, anterior end of right cord and ventricular band and anterior portion of thyroid cartilage.

Carcinoma of right cord, extending to anterior commissure. Excised specimen includes entire arytenoid, anterior third of left cord and ventricular band and portion of underlying cartilage.

Partial laryngectomy for carcinoma of left cord including anterior end of right cord and ventricular band and most of left thyroid ala. Recurrence in ant. commissure after 10 mo. See next specimen.

Laryngectomy for recurrent carcinoma following partial laryngectomy (see previous specimen). Area of previous skin graft along left side of glottis is well shown.



Carcinoma of left cord; carcinoma in situ of right cord. Specimen includes entire cord and ventricle, with portion of ventricular band on each side, along with anterior portion of thyroid ala.

Carcinoma of laryngeal surface of epiglottis, excised by partial horizontal laryngectomy (Alonso) with secondary closure of pharyngostoma. Living and well after three years.

Carcinoma of tip of epiglottis, recurrent following X-ray therapy 8 months previously. Excision by partial horizontal laryngectomy (Alonso technic).

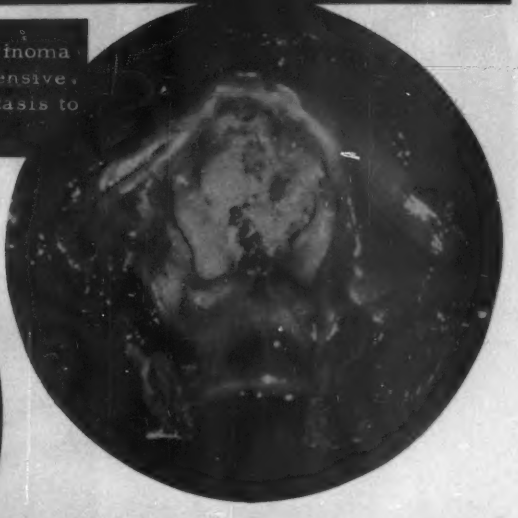
Extensive glottic and supraglottic carcinoma in a patient with advanced bilateral pulmonary tuberculosis. No microscopic evidence of tuberculosis in surgical specimen.

Unusual posterior subglottic origin of carcinoma. Larynx has been opened anteriorly to show lesion. Cords not grossly or microscopically involved.

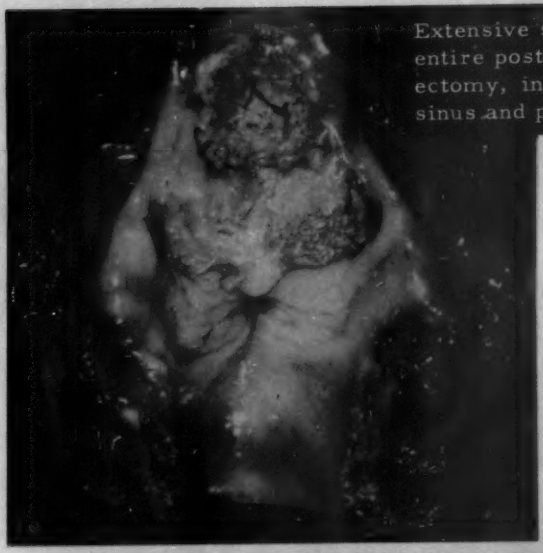
Large carcinoma of epiglottis and aryepiglottic fold. Laryngectomy, including pyriform sinus, vallecular region and portion of base of tongue.

Lesion of left pyriform sinus - lateral wall of larynx. Left cord excised by laryngofissure 5 years previously; no recurrence on interior of larynx.

Laryngectomy for undifferentiated carcinoma previously treated by irradiation. Extensive supraglottic lesion. Subsequent metastasis to lung and brain.



Carcinoma of right vocal cord with fixation and beginning subglottic extension. Laryngectomy without neck dissection (no palpable nodes).

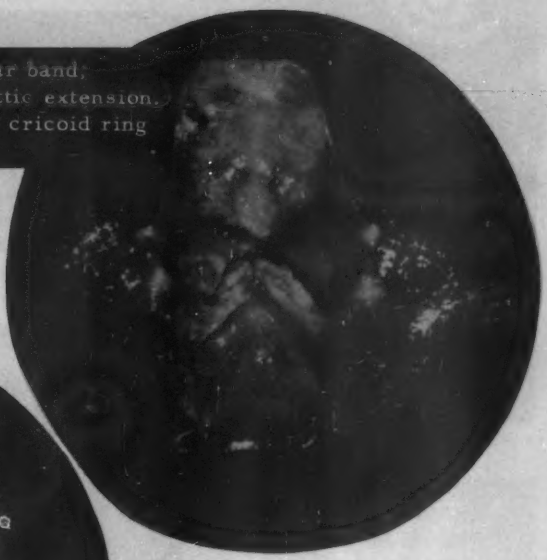


Extensive supraglottic carcinoma, involving entire posterior surface of epiglottis. Laryngectomy, including large portion of each pyriform sinus and pre-epiglottic space.

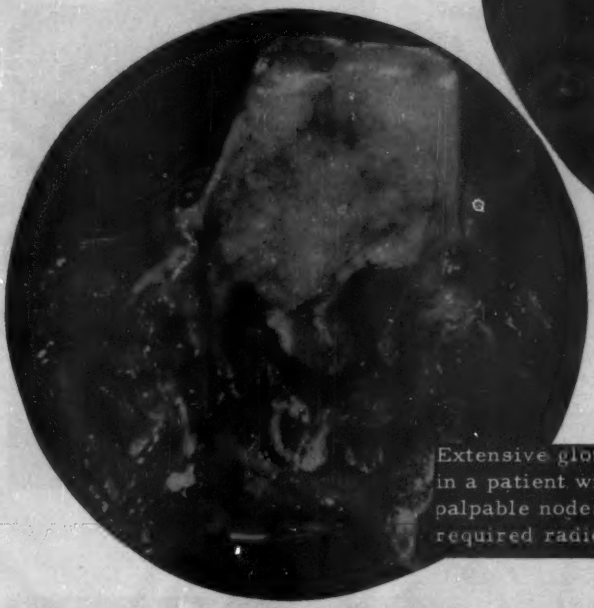
Laryngectomy for carcinoma recurrent one year following laryngofissure. Inner opening of tracheotomic fistula at lower border of specimen.

Moderately extensive carcinoma of left vocal cord, involving ventricle and ventricular band. Treatment by narrow-field laryngectomy.

Extensive carcinoma of supraglottic larynx with infiltration and necrosis of vallecular region and base of tongue. · Laryngectomy and resection of large portion of tongue.



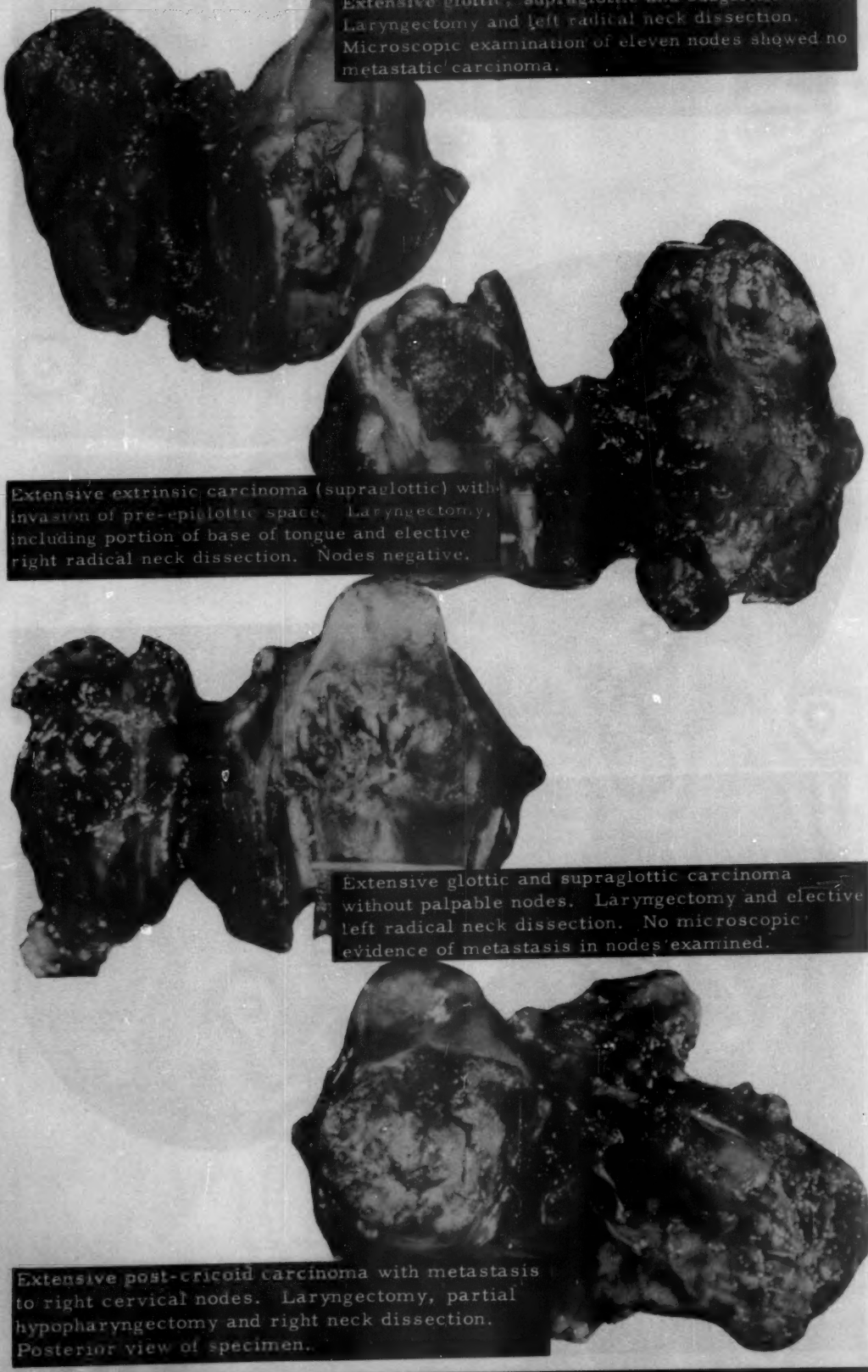
Carcinoma of left cord and ventricular band; leukoplakia of right cord. No subglottic extension. Laryngectomy, conserving portion of cricoid ring for support of stoma.



Extensive glottic and supraglottic carcinoma in a patient with untreated tertiary lues. No palpable nodes at time of operation, but later required radical neck dissection for metastasis.

Well differentiated carcinoma of right cord. Treatment by partial laryngectomy not thought advisable because of fixation and beginning subglottic extension.

Extensive supraglottic carcinoma with invasion of preepiglottic space and palpable metastasis. Laryngectomy including portion of base of tongue and left radical neck dissection.



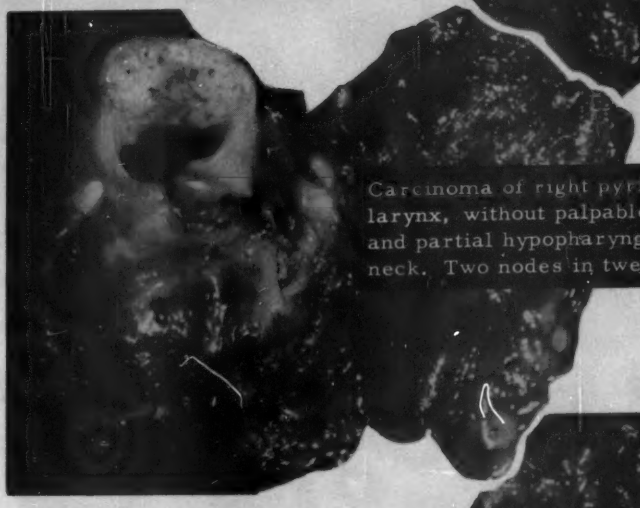
Extensive glottic, supraglottic and subglottic lesion. Laryngectomy and left radical neck dissection. Microscopic examination of eleven nodes showed no metastatic carcinoma.

Extensive extrinsic carcinoma (supraglottic) with invasion of pre-epiglottic space. Laryngectomy, including portion of base of tongue and elective right radical neck dissection. Nodes negative.

Extensive glottic and supraglottic carcinoma without palpable nodes. Laryngectomy and elective left radical neck dissection. No microscopic evidence of metastasis in nodes examined.

Extensive post-cricoid carcinoma with metastasis to right cervical nodes. Laryngectomy, partial hypopharyngectomy and right neck dissection. Posterior view of specimen.

Extensive carcinoma of left pyriform sinus and extrinsic larynx without palpable metastasis. Laryngectomy, partial hypopharyngectomy and left radical neck dissection. Metastasis microscopically in cervical nodes.

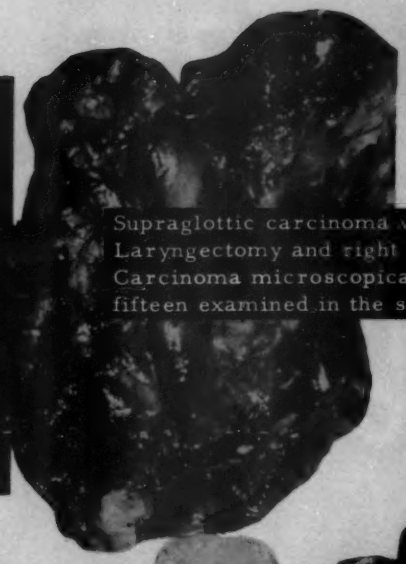


Carcinoma of right pyriform sinus and extrinsic larynx, without palpable metastasis. Laryngectomy and partial hypopharyngectomy with right radical neck. Two nodes in twelve showed metastatic tumor.

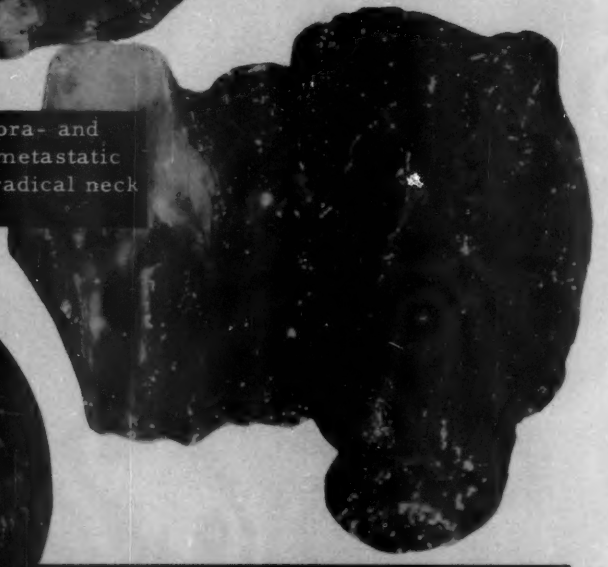
Extensive carcinoma of left supraglottic larynx with palpable nodes, positive on section. Two months later, right radical neck dissection for metastasis developing post-operatively. Subsequent lung metastasis.

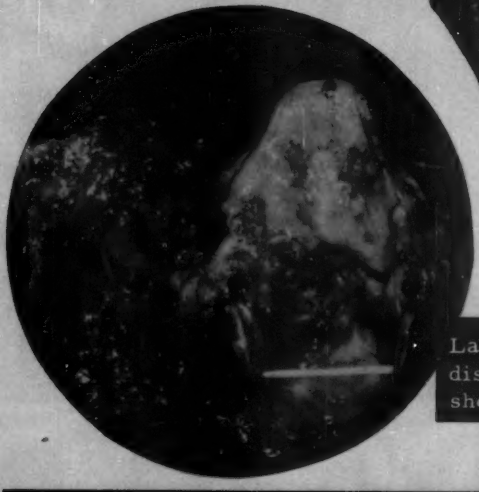
Extensive supraglottic carcinoma. Laryngectomy with excision of portion of base of tongue and elective left radical neck dissection. Surgical specimen showed no metastatic carcinoma in cervical nodes.

Supraglottic carcinoma with palpable metastasis. Laryngectomy and right radical neck dissection. Carcinoma microscopically in one node of the fifteen examined in the surgical specimen.



Carcinoma of right cord, with supra- and sub-glottic infiltration. Palpable metastatic nodes. Laryngectomy and right radical neck dissection.





Laryngectomy and elective left radical neck dissection. Histologic study of specimen showed no involvement of nodes.

Extensive bilateral intrinsic carcinoma. Laryngectomy and elective right radical neck; nodes negative. Subsequent metastasis to left cervical nodes treated by radical neck dissection.

Unusually extensive glottic, supraglottic and subglottic carcinoma with palpable metastasis and previous tracheotomy. Laryngectomy and right radical neck dissection.

Glottic and subglottic carcinoma without palpable nodes pre-operatively. Nodes palpable along right carotid sheath on exploration. Laryngectomy and right radical neck. Nodes positive on section.

Glottic and supraglottic lesion with palpable metastasis. Laryngectomy and left radical neck dissection.

Glottic and subglottic carcinoma. Laryngectomy and right radical neck dissection. Suspicion of palpable metastatic nodes not confirmed by microscopic examination of surgical specimen.

INDICATIONS for TOTAL LARYNGECTOMY

1. CANCER OF VOCAL CORD reaching posterior end, producing fixation or extending subglottically (narrow-field laryngectomy)
2. CANCER OF VENTRICULAR BAND OF VENTRICLE (wide field laryngectomy with or without simultaneous radical neck dissection)
3. CANCER OF EPIGLOTTIS (except very early cases with involvement of only the crest.)
4. CANCER OF LARYNX with palpable cervical metastasis (laryngectomy combined with simultaneous - radical neck dissection)

INDICATIONS for IRRADIATION

1. CANCER OF VOCAL CORD too extensive for laryngofissure (involving posterior extremity) but without fixation
2. CANCER OF THE EPIGLOTIS, especially when of undifferentiated type.
3. CANCER OF LARYNX considered inoperable because of extent of involvement of pharynx or base of tongue.
4. CANCER OF LARYNX in which laryngectomy is contraindicated by physical condition or temperament of patient, or is refused.

TREATMENT of RECURRENCE and/or METASTASIS FOLLOWING LARYNGECTOMY

	TOTAL CASES	DETERMINATE CASES	DEAD OF DISEASE	5 YEAR SURVIVAL
RESECTION OF RECURRENCE	5	4	2	2
RESECTION PLUS X-RAY	3	3	2	1
RADICAL NECK DISSECTION	3	3	2	1
RADICAL NECK PLUS IRRADIATION	9	9	7	2
IRRADIATION ALONE	28	27	26	1
UNTREATED or TREATED ELSWHERE	15	15	15	0
TOTALS	63	61	54	7

FIVE YEAR SURVIVAL RATE (Salvage) 7/61 = 11%

TREATMENT OF RECURRENCE FOLLOWING IRRADIATION

	TOTAL CASES	DETERMINATE CASES	DEAD of DISEASE	5 YEAR SURVIVALS	
Further Irradiation	19	19	14	5	26%
Partial Laryngectomy	3	3	0	3	100%
Laryngectomy	6	5	1	4	80%
Laryngectomy-Radical Neck	4	4	2	2	50%
No Treatment	7	7	7	0	
TOTALS	39	38	24	14	37%

FIVE YEAR SURVIVAL RATE (Salvage) 14/38 = 37%

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